THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—13TH YEAR. SYDNEY: SATURDAY, OCTOBER 23, 1926. No. 17.

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THE PREVENTION OF TUBERCULOSIS.[1]

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IT is perhaps hardly necessary at the outset of this paper to say that the prevention of tuberculosis involves two distinct problems—the prevention of the spread of bovine tuberculosis to man and the prevention of the spread of human tuberculosis in man. The former is largely the cause of bone and joint tuberculosis in children (but not of tuberculous meningitis which is really the most prominent evidence of our acute miliary tuberculosis and is generally of human origin). Important as it is, its ravages are very small in proportion to those of pulmonary tuberculosis, which is always of human origin. For this reason and because the control of bovine tuberculosis is entirely a question of milk supply which is the responsibility of dairy experts, I do not propose to touch on it in the limited time at my disposal, but to concentrate on certain aspects of the problem of prevention of human tuberculosis. Naturally some aspects only can be touched upon. In order to make the discussion as practical as possible, I shall deal mainly with local conditions, as they constitute our particular problem.

One other preliminary question must be touched on. Great emphasis has been laid of recent years on the distinction between tuberculous infection and tuberculous disease. We are often told that we have all been infected, but only a comparatively few suffer and die from tuberculous disease, and the term tuberculosis is usually confined to the latter condition. By law, for instance, we are supposed to notify anyone suffering from pulmonary tuberculosis, yet no one dreams of notifying all victims of tuberculous infections. Pathologists maintain that this tuberculous infection which has been proved to be present in 90% of people in Europe and America and 60% in Australia who reach adult

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 29, 1926.

life, protects us from a fresh infection in adult life. Extremists hold that all cases of active tuberculosis which show themselves in adult life, are due to the spread or lighting up of such an old infection. On this assumption one's main object tonight should be to discuss means of building up the resistance of the infected individual.

I pass by any discussion of this point which would take up the whole evening, and shall simply say that clinicians recognize and radiography of recent years has emphasized the fact that cases of pulmonary tuberculosis seen in adults do often arise in this way, the fresh activity of the latent lesion following on exhausting lactation or exhausting chronic disease or lighting up after the heat of a trying summer or after an infection like influenza or measles. On the other hand they cannot overlook the frequency with which they see active tuberculosis arise in adolescents or young adults after more or less recent exposure to massive infection. If these are all coincidences, they are extraordinarily frequent.

The whole question seems to resolve itself into the relation between the amount of protection given by the first infection and the massiveness of the fresh infection to which the same individual may be exposed; but we have no means of measuring either of these. On this account emphasis needs to be laid as much on the lessening of infection as on the increasing of resistance. While at present it may be a counsel of perfection to hope for complete prevention of infection, to dwell only on the subject of building up resistance is a counsel of despair.

In this State, indeed, it would seem that at present the lessening of infection needs to be the more emphasized. For in this State during the last twenty-five years more than three hundred deaths each year have occurred from pulmonary tuberculosis. It was with something of a shock that we learnt at the last Melbourne Congress that Adelaide had the highest death rate from pulmonary tuberculosis of all the capital cities of Australia.

City.	Pulmonary Tuberculosis.			Tuberculosis other than Pulmonary.		
	Mean Mortality 1900-1901.	Mean Mortality 1920-1921.	Percentage Reduction.	Mean Mortality 1900-1901.	Mean Mortality 1920-1921.	Percentage Reduction.
Brisbane	122	70	42	37	3·5	90·5
Sydney	108	50	54	18·5	11	40·5
Melbourne	140	86	45	43·5	19	56·3
Hobart	97	80	18	36·5	16	56·2
Adelaide	122	101	18	24	16·5	31·3
Perth	110	62	43	21	15·5	36·2

Attempts have been made to question these figures, but their accuracy is borne out by the graph which I show you (Appendix I.), which demonstrates, (i.) that the death rate from pulmonary tuberculosis for South Australia is at present higher than that for the whole of Australia; (ii.) that while it has fallen in our State during the last twenty-five years, it has not fallen in the same proportion as that for Australia as a whole. For twenty-five years ago it was below the general rate for Australia, whereas now it is above that rate.

No clearer example of the uselessness of notification without well planned administrative measures could be given, for this State was the first to introduce notification of pulmonary tuberculosis over twenty-five years ago.

The standard of living has appreciably improved during these years. There is undoubtedly greater aggregation of people in the city, but on the whole much more open air living. There is certainly not so much overcrowding here as in Melbourne and Sydney. Moreover, Dr. F. E. Wynne[1] has shown that in England although "overcrowding exists now to an extent probably unequalled since the 'forties of the last century when hunger was the real scythe of death," the death rate from tuberculosis continues to fall there in spite of the overcrowding. There are evidently other powerful factors at work, of which the control or lack of control of opportunities for infection is not the least.

If three hundred adults die annually in this State of pulmonary tuberculosis, on the modern estimate of ten active cases alive for every death, that would mean that there are three thousand cases in the State. When we consider the paucity of hospital accommodation for these persons—

Kalyra (for early cases) .. 50 beds
Bedford Park .. .. 59 beds
Consumptive Block .. .. 64 beds
and about twenty patients usually present in Adelaide Hospital,

we must realize what opportunities for infection must exist, through lack of control of cases outside these institutions.

Certain evidence suggests that this infection takes place rather later in life than in European countries. The graph (Appendix II.) taken from New York figures shows that in America the great rise in infection takes place between two and six, as soon as the child leaves his own back yard, so that by the age of six some 60% of children are infected.

Investigations in this State a few years ago showed, however, one hundred and ten patients in the Children's Hospital from all diseases, who ranged in age from under one year to thirteen, only 18·2% gave positive von Pirquet reactions and in one hundred and forty-three children of similar age tested by Dr. Beare only 23% gave a positive von Pirquet reaction. There were practically no "positives" under one year of age. In contrast with this about 60% of healthy school children in Victoria gave positive reactions, whereas in Children's Hospital patients in Melbourne as in Adelaide, only 20% gave "positive" reactions. Dr. Penfold suggests that other illnesses besides acute tuberculosis suppress the von Pirquet reaction. This research undoubtedly needs to be prosecuted on a larger scale. It would be interesting, now that the Education Department has an augmented staff of school medical inspectors, for such a piece of research to be done amongst a large number of school children, as has been done with the Schick test.

Evidence is daily seen in the medical wards that infection chiefly occurs amongst family and other close contacts of patients with open lesions, often before the latter are recognized. The figures of the Royal Prince Alfred Hospital Clinic in Sydney showed that 57% of contacts gave a positive von Pirquet reaction.

It is easy for us to say that the infected individual must go to a sanatorium and be taught how to disinfect all sputum and so on. This overlooks the two great sources of danger at present, the missed cases and the advanced cases that are the sources of massive infection. The removal of the first danger is largely our responsibility as members of the medical profession. I have been surprised and depressed of recent years at the frequency with which patients admitted to my wards have been told by outside practitioners that they were suffering from bronchitis. One of these patients had had two previous attacks of pleurisy with effusion; in another one who was a railway porter, the chest trouble had been going on for nine or ten years, during which he had been stationed at several different railway stations, yet the first examination of sputum by a student showed the presence of numerous tubercle bacilli. The rule that was laid down when I was a student still holds good, namely that a one-sided bronchitis never occurs and that persistent cough and expectoration should never pass without repeated examinations of the sputum for tubercle bacilli.

I have been more surprised at the frequency with which medical practitioners continue to regard lightly, attacks of hæmoptysis in young adults. Patients come into the ward with advanced signs in their lungs, who have previously consulted their medical attendant for hæmoptysis and have been told they could return to work in a day or two. Yet authorities were teaching even before I was a student that hæmoptysis in a young adult should always be regarded seriously. Just as disquieting is the number of cases that are seen with active pulmonary tuberculosis, who gave a history of pleurisy with effusion two or three years previously, who never had it impressed on them that 75% of such cases are tubercular in origin and can be arrested in that stage.

When these examples of carelessness exist, it is perhaps waste of time for me to insist on the need of careful and continuous observation of young adults with unexplained dyspepsia, anæmia or loss of weight. But I think that this class of case is often more thoroughly investigated than those to which I have referred. The average medical mind is very apt to label things and to be content as soon as he finds a label, but to investigate more thoroughly if he cannot find one.

I have emphasized this point, because it is very little use our leaders clamouring for increased facilities for early diagnosis when we do not avail ourselves of the facilities which we possess. These cases, recognized early, would be the proper applicants for sanatorium treatment, but it is no use our agitating for more sanatorium accommodation if we are missing these early cases. Not merely are they losing their opportunity for arrest of disease, but they are infecting others.

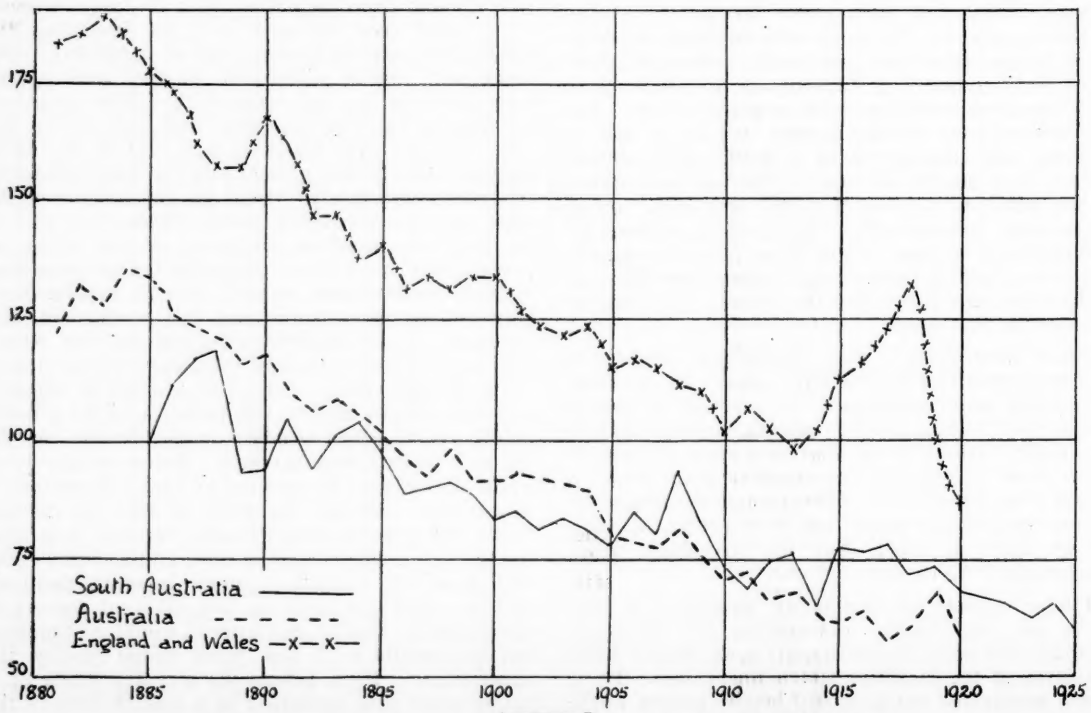
I am glad to find that the more recent graduates are much more careful over these conditions of which I am speaking. Yet with the absence of any coordinated attack on the problem throughout the State their experience soon tends to dishearten them. I can best demonstrate this by the following extract from a letter recently received by me from one of our keenest young country practitioners. The letter accompanied the patient whom he sent down. After discussing the difficulties in the matter of tuberculosis, he says: "Now take this chap. He was told after a very uneventful course at Kalyra that he was practically a healed case. He had rested about ten months when I saw him and was always afebrile with pulse 70 to 86. He put on weight, felt fit and rested quite conscientiously. Then came the trial for me. He confronted me with his wife and kiddy; told me of their hardships, of money borrowed from parents to keep going and of fast disappearing resources. He had had nine months' spell, was fit and well apparently and could get an outdoor clerical job, free from all strain. He was obviously fretting at inactivity and financial stress; he would at any rate be happy earning a little money; and he was apparently well, with no demonstrable signs of activity. Well, I relented. Yet three weeks ago he had another small hæmoptysis after only four months' light work in the open air and we are worse off now than before apparently." By the irony of fate I learnt from the patient that the "light clerical work" had been changed to a job involving the pushing for several hours a day of a railway truck laden with tins of petrol and the next week a man was admitted to my ward with fairly advanced pulmonary tuberculosis, who had been living for some months with his wife in a tent in the same railway camp as this patient and had been spreading tubercle bacilli freely. Great are the benefits of the open air! Is not this a perfect illustration (i.) of the economic problems hindering all our attack on tuberculosis; (ii.) of the necessity for the provision of some employment colony scheme, intermediate between the sanatorium and full work; (iii.) of the need of an administrative scheme for State-wide supervision of cases of pulmonary tuberculosis?

To advocate these reforms as matters of urgency is a duty we owe alike to our patients, to the general public and to other members of our Branch, as the letter I quoted demonstrates. There should be a whole time expert at the head of such a department, who should correlate the work of different institutions and general practitioners, arrange a scheme for the better supervision of open and arrested cases and generally stimulate public interest, administrative action and research into different aspects of the problem. Such a department would cost money, but its results in a few years would justify the expenditure. Even before the war the death of a young adult was estimated as a loss of £500 to the community. On this low estimate for present con-

ditions, the deaths in this State last year meant an economic loss of £150,000, quite apart from the loss due to the invalidity of so many more sufferers.

The other great danger of the spread of infection arises from the advanced cases of the disease. In numbers of patients admitted to the wards, one can definitely trace such direct infection. The first line of defence against this danger is the provision of a sufficient number of beds for patients with advanced disease, so that where home accommodation is definitely insufficient or where the patient or relatives refuse to take precautions, segregation may be ordered. About fifteen years ago a bill was introduced into the local Parliament directing that in cases where two medical practitioners certified there was this neglect of precautions, the patient might be summoned before a stipendiary magistrate and if that official were satisfied that such neglect of precautions existed, he might make an order for segregation. I have always thought it was a pity that this bill was not proceeded with. It safeguarded equally the rights of the individual sufferer and the individual contact. But such a law would be useless until we provide ample accommodation for these patients. In the travels of the Royal Commission on Health through Australia last year we found that no State had sufficient beds for these patients. The present position is absurd in that these patients receive Commonwealth pensions, the sole result of which is the raising up of a fresh generation of pensioners. The evil is intensified by some regulation that such patients who enter institutions, have their pensions lessened. From a preventive aspect, it would be far wiser to increase the pension to those who enter institutions and to pay pensions to their dependants. Here again the economic side comes to the front.

The immediate family contacts of these missed and advanced cases are the prospective patients of the future. The only method of dealing with these contacts satisfactorily as regards prevention is by the establishment of a tuberculosis dispensary or clinic in every large centre. To it should be transferred all persons with doubtful signs, who should be kept under regular observation and cannot pay for it, also those with arrested disease discharged from sanatoria for similar prolonged observation. Nurses from such a clinic should visit the homes of such patients and the homes of patients with advanced disease, should see that all proper precautions against infection are being carried out and should arrange for family contacts to come to such clinics for examination in regard to the existence of infection by physical, serological and radiological methods. Such a clinic is best established in connexion with a general hospital, but should be arranged so that general practitioners may avail themselves of these opportunities of special examination for any of their own patients and contacts. For I am certain that prevention will never be advanced until the general practitioner is brought in as an active participator in the scheme. His interest needs to be quickened by the provision

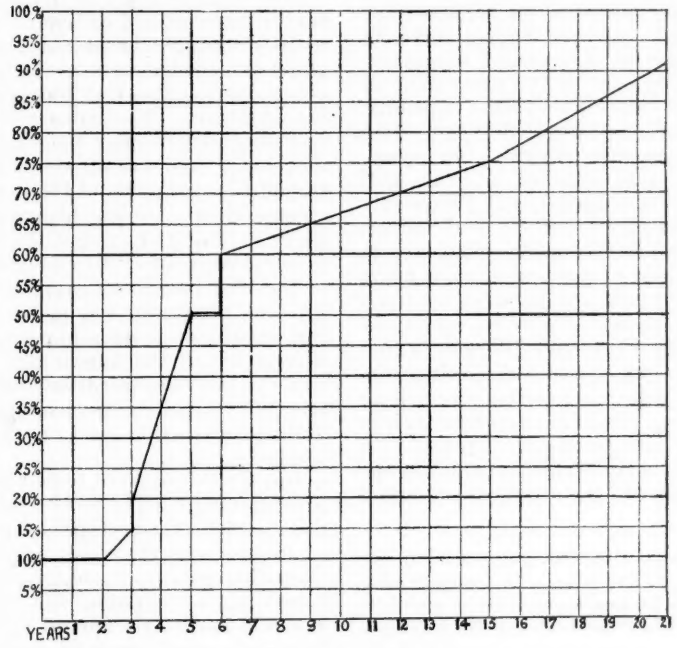


Appendix I.

Showing Death Rates from Pulmonary Tuberculosis per Hundred Thousand of Mean Population.

of these facilities. One effect of notification has been to instil into his head the idea that prevention of further infection is the job of the administrative authorities. For that reason he needs to be brought in as part of the administration.

All these points that I have raised, the need for research, for early diagnosis, for intermediate occupational colonies, for segregation for patients with advanced disease and for thorough and frequent observation of close contacts, emphasize the need for the creation in our State of a special department of tuberculosis in connexion with our State Department of Health. Before the war the Central Board of Health had an arrangement by which nurses periodically visited the homes of notified patients. This was abandoned, I think at the beginning of the war. But with our increased knowledge much more than this is required. It will not be obtained unless we, as a Branch, persistently urge it and I hope that this meeting will see the beginning of some such movement on our part. The Victorian Department of Health presented to the Royal Commission on Health an outline of their scheme, though they have as yet only got a very small part of it in operation. I present to you the schematic outline of their proposals.



Appendix II.
Showing Tuberculous Infection in Human Beings soon after Birth until Entrance into Adult Life.

All these methods are, however, but stopgaps. It is yearly becoming more evident that the main hope of control of all infections which are spread by the secretions of the mouth and nose, must be by some system of protective inoculation of those exposed to infection. After one hundred years' experience of the truth of this for smallpox, the method has of recent years been extended to the prevention of diphtheria, whooping cough and scarlet fever and possibly influenza. There are, at last, possibilities that some such method may be soon available for pulmonary tuberculosis. Calmette claims after thirteen years' work with successive cultures of what was originally a very virulent bacillus of bovine tuberculosis, that he has prepared a vaccine of living modified organism which would not set up tuberculosis, but would confer immunity after a course of inoculation. After proving it to be harmless to animals, in 1922 he vaccinated one hundred and seventy-eight infants born of tuberculous mothers, many of them living in close contact with their infective mothers. Past experience has shown that normally 25% of such children (or one in four) would die of tuberculosis infection before they were a year old. The tuberculous mortality for those vaccinated in 1922 was nil. Of the one hundred and seventy-eight vaccinated, fifteen had died up to June, 1925, when these results were published, but none showed signs of tuberculous infection. The mortality from all diseases in this group was 8·4%, the average mortality in France of infants under one year is 12½%. Such a method, if these successes are confirmed by further experience, is along the lines of true prevention. It differs as much from our present clumsy methods as vaccination against smallpox differs from the inoculation which was previously in vogue. It is interesting to know that cultures have been obtained from Calmette for Australia by the Commonwealth Serum Laboratories and it is to be hoped that we shall soon be able to gain experience of this new method in our own, as well as the other States.

Reference.

(1) F. E. Wynne: "Overcrowding and Epidemic Disease," *Proceedings of the Royal Society of Medicine*, March, 1925, page 29.

SOME NOTES ON THE SPECIFIC TREATMENT OF PULMONARY TUBERCULOSIS WITH REFERENCE TO THE PATHOLOGICAL TYPES AND THEIR DIAGNOSIS.[1]

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As different types of tuberculosis call for different methods of treatment, I want to spend a little time in the consideration of their pathological grouping and the one I find most useful is that of Ranke slightly modified by Neumann.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 29, 1926.

Type I. Tuberculosis.

Type I. is the simple primary complex in which there is a tuberculous nodule in the lung and an infection spreading along the lymphatics around the bronchi to the corresponding lymphatic gland. This primary complex is common in children in whom the primary nodule is usually found towards the apex of the lung, but may be close to the diaphragm or to the mediastinal pleura. In adults it is usually situated five to six and a quarter centimetres (an inch to an inch and a half) below the apex of the lobe. In this first type there is no extension into the blood stream nor has the bronchial tree been affected. It is essentially a closed type of tuberculosis limited to the primary nodule, the lymphatics and the lymph glands. The symptoms are due to the enlarged glands in the mediastinum or to their pressure on neighbouring structures, for example, on the vagus nerve in which case there may be symptoms suggesting ulceration of the stomach or of the duodenum or even inflammation of the vermiform appendix. If the primary focus is situated very close to the right diaphragmatic pleura, the symptoms might suggest inflammation of the gall bladder or ulceration of the duodenum. Frequently the right trapezius muscle is painful and tender. If the primary focus is near the mediastinal pleura, the child might complain of pain with each heart beat. The physical findings are those of enlarged bronchial glands with Kramer's dulness on one or both sides of the vertebral column. D'Espine's sign is usually present, the whispered voice being conducted down to the spine of the sixth or seventh thoracic vertebra. Very often the vertebral spines are tender when they are percussed. Owing to a neuritis of the phrenic nerve Mussey's points in the neck or in the upper part of the abdomen are found to be tender.

The primary focus is situated close under the pleura on the surface of the lung. It consists of a rapidly caseating broncho-pneumonic focus which is sharply delimited from the rest of the lung. It is always accompanied by a somewhat larger caseous focus in the related lymphatic gland. The primary infections of childhood are capable of producing a typical form of scarring and not only do they calcify, but they have a definite tendency to ossification. The primary focus is usually single. The reinfection in contrast to the primary infection may be multiple. It occurs essentially near the apex of the lung, less commonly in other situations. Usually it is situated in the substance of the lung and when it heals, it causes contraction of the surface. It is especially characteristic that the related lymphatic gland is free from infection.

The reaction to the primary infection is an anaphylactic one in that there is a large outpouring of cells. When it heals a certain degree of immunity is produced, so that the reinfection occurs in a partially immunized individual. Those who have not had this primary infection in childhood, cannot rely on any degree of immunity when they are brought into contact with an infection at the time of puberty or early adult life. The result is that they succumb to a very rapidly spreading form of disease which again might be called an anaphylactic reaction. This was very well seen when the black troops were taken across in the army of occupation. Thus we must remember that with the prevention of the primary infection in childhood, as Aschoff has pointed out, the adult is sent forth into life unprotected against this disease and it will attack him with infinitely greater virulence than the usual infection in childhood. And this is the reason for Calmette's active immunization of infants.

Type II. Tuberculosis.

Type II. is the pubertive type of pulmonary tuberculosis or galloping consumption. In it the tubercle bacilli have gained entrance to the blood stream usually through the breaking down of a lymphatic gland and the entrance of the bacilli to an ulcerated blood vessel. Also they have gained entrance to the bronchial tree. The primary focus has broken down and ulcerated through the wall of an alveolus or of a bronchus and by aspiration the bacilli have been spread to other parts of the lung, producing patches of tuberculous broncho-pneumonia. A lot of hyperæmia is produced around the alveoli which become filled with proliferated endothelial cells. This part of the lung now looks rather like a piece of spleen; it is red and soft and moist and the French call the condition a splenic pneumonia. Owing to a diminution of the hyperæmia the endothelial cells undergo fatty degeneration and the condition is then one of gelatinous pneumonia. Lastly the cells undergo caseation and a caseous pneumonia is produced.

During the stage of hyperæmia or congestion the process may resolve or it may go on to gelatinous pneumonia. This gelatinous pneumonia may go on to fibrosis and thus heal or the cells may caseate. Caseation goes on to destruction of tissue with the formation of cavities. In this second type the tubercle bacilli are spread through the lung tissue by three routes; firstly by the blood stream, secondly by the lymphatics and thirdly by the bronchial tree. This type is found in young people. The majority of the cavities are in the front of the chest, especially below the middle third of the clavicle. Often these patients are seen with the symptoms and physical findings of an ordinary pneumonia, but I would like to call attention to the following points which assist in the differential diagnosis:

(i.) There is no increase of fibrin in the blood.
(ii.) The chlorides in the urine are not diminished.
(iii.) There is no rapid sedimentation of red blood corpuscles.
(iv.) The number of leucocytes is not increased.

These cases run a very rapid course and the patients die in from a few weeks to a few months. Besides the local findings in the lungs there are usually signs of spread out through the blood stream to distant organs such as the spleen which is en-

larged and soft with a rounded edge, or to joints, epididymis, kidneys or meninges.

Type III. Tuberculosis.

Type III. is the most important and includes most of the patients seen in practice. This type is known as the proliferating primary complex, the proliferation involving the lymphatic and blood streams, but not involving the bronchial tree to any appreciable extent. As evidence of the hæmorrhagic spread of the bacilli we find distant organs involved, for example, a tuberculous lesion of a bone, of a kidney, of a suprarenal capsule or of an eye. The arteries are usually rigid, though the blood pressure is low and the spleen is enlarged and firm with a sharp border.

There are several subgroups of this third type and I shall refer to them quite briefly.

In the simple proliferating primary complex group there are enlarged bronchial glands and evidence of hæmorrhagic proliferation in rigid arteries, hard, sharp bordered spleen and lesions of distant organs.

Patients in the typhoid group have prolonged fever, but there are no physical findings in the chest which are distinctive, the Widal test yields no reaction and there are no typhoid bacilli in the blood nor in the stools. The sputum does not contain tubercle bacilli. These patients generally get well in that the temperature settles down, but they do not make a good convalescence, for they do not put on weight nor do they regain their appetite. In a few months time they are found to have definite lesions in their lungs.

Polyserositis (the third group) can be active or healed. There may have been effusions into all the serous sacs—pleural, pericardial and peritoneal and even the joint cavities may have been involved. There may be evidence of past trouble in these cavities, such as thickening and adhesion of the pleura, pericardial adhesions or even obliteration of the pericardial sac. The peritoneum might be thickened over the liver or spleen or the mesentery may have undergone shrinkage.

A fourth group is called *tuberculosis fibrosa diffusa* and in this group there are scattered miliary tubercles throughout the lungs. These miliary tubercles may not be visible with the X rays nor may they give rise to much in the way of physical signs. But a compensatory emphysema is produced in these cases—we find low-standing lung borders with the heart covered with the emphysematous lungs so that there is very little, if any, superficial cardiac dulness. Thus the physical findings would be emphysematous lungs in a young person with perhaps small scattered patches of pleural friction, rigid arteries and a palpable spleen with a hard sharp edge.

The fifth and last group is known as *tuberculosis fibrosa densa*. These patients usually have dense pleural thickenings over both apices with perhaps an adhesion at one or other base. It is common in patients with surgical tuberculosis, for example, of the spine or of the knee. Also it is common in emphysematous old people in whom one would expect to find very wide Krönig's fields but in whom these fields are found to be very narrow. Both *tuberculosis fibrosa densa* and *diffusa* can go on to ulceration and cavity formation.

Patients who have or have had several serous sacs involved, frequently complain of trouble in their eyes or of muscular rheumatism. Many cases of episcleritis, scleritis, iritis, chorioiditis and iridocyclitis are now known to be due to the tubercle bacillus. Previously they were regarded as rheumatic infections but in reality they are due to what the Germans call *"unspezifische Tuberkulose."* The lesions are found to consist of granulation tissue without definite epithelioid or giant cell systems, but when injected into guinea pigs or by the use of "Antiformin" in frozen sections tubercle bacilli have been demonstrated. Some of these patients complain only of muscular rheumatism which is due to the circulating toxins or to the presence of the tubercle bacillus itself. This condition has been described by Poncet and is known as Poncet's rheumatism.

Type IV. Tuberculosis.

Type IV. is the last type and in this the proliferation is almost entirely by the broncial tree. Hæmorrhagic proliferation and spread along the lymphatics with enlarged glands are not evident or at least are not prominent features. Whereas in Type II. the cavities were most pronounced in the front of the chest, in this fourth group they are most evident when the back of the chest is being examined. There is no evidence of hæmorrhagic proliferation and the only lesions which are produced apart from those in the lungs and pleura, are tuberculosis of the larynx and of the intestines. These lesions are due to the expectoration and to the swallowing of infected sputum.

Summary of Types of Tuberculosis.

To summarize the main characteristics of these four types:

Type I., simple primary complex has no tendency to spread. The glands are enlarged. The arteries are not rigid nor is the spleen enlarged. Symptoms and signs are due to enlarged bronchial glands.

Type II., pubertive type of phthisis, progresses by the lymphatics, by the blood stream and by the bronchial tree. The glands are enlarged. The spleen is not affected if the condition is acute. The cavities are most pronounced in the front of the chest.

Type III., proliferating primary complex, is characterized by enlarged glands. The spleen is palpable and has a hard sharp edge. The arteries are thickened. Distant organs are affected through the proliferation by the blood stream.

Type IV., fibro-caseous cavernous tuberculosis, has no enlarged glands; the spleen is large and has a rounded border and is not hard. The arteries are soft. The cavities are most common at the back of the chest.

I have spent so much time on the description of these four types because each one demands a different form of treatment.

Type I. responds very well to treatment with tuberculin ointment.

Tuberculin.

Before proceeding any further I should like to discuss quite briefly the use of tuberculin in diagnosis and treatment.

For the subcutaneous tuberculin test either old tuberculin or the albumose free preparation (T.A.F.) which is standardized to the same strength, may be used. Rivière considers a convenient and safe dosage to follow is the series 0·2, 1·0, 5·0 and 10·0 cubic millimetre and half this dosage for children. The injections should be given at intervals of forty-eight or seventy-two hours and it is best that they be given in the early morning or late at night, as the reaction does not appear for six or eight hours. A three or four hour temperature chart should be kept for three days before the test and after the injection the temperature should be taken every two hours while the patient is awake. Every rise in temperature must be looked on as a reaction and a larger dose must not be given. If this rule be followed, dangerous focal reactions will be avoided.

Rivière gives several definite contraindications to the use of this test:

(i.) Fever. Koch wrote: "Patients with temperatures over 37° C. in the axilla are unsuitable for the diagnostic administration of tuberculin and should under no circumstances be submitted to the tuberculin test."

(ii.) Obvious phthisis. Here the test is needless and dangerous.

(iii.) Recent hæmoptysis.

(iv.) Other contraindications are suspicion of miliary tuberculosis, recent severe illnesses, serious diseases like diabetes in the very young, advanced arteriosclerosis, kidney disease, myocarditis and epilepsy.

Bandelier and Röpke give practically the same contraindications. The subcutaneous tuberculin reaction is made up of the following four components: (*a*) The rise of temperature (febrile reaction); (*b*) disturbance of physical well being (general reaction); (*c*) local inflammatory reaction at the site of injection (needle track reaction); (*d*) the inflammatory reaction at the focus of the disease with increase of cough or sputum or physical signs or of all three (focal reaction).

The test depends on tissue response and so may be expected to fail in acute illnesses, in the moribund and in advanced pulmonary tuberculosis. Also a tuberculous patient may be tolerant as a result either of previous tuberculin treatment or in some cases of successful autoinoculation. In cases of early and slight disease an absence of reaction furnishes strong evidence against the presence of active tuberculosis. The interpretation of a positive response is difficult. It can be stated that a prompt reaction to a small dose generally denotes recent and active disease. In the absence of physical signs or other evidence the patient should be kept under observation for a certain period. A reaction to larger doses proves only the presence of former infection.

A focal reaction is of very definite value, as it indicates not only the position of the disease, but also the tuberculous nature of this and is, moreover, presumptive evidence that the focus is active or may become so. The focal reaction provides the one source of danger in the use of this test. Its appearance is definite proof of the presence of the tubercle bacillus. Bandelier and Röpke state that:

> After childhood it is the sovereign diagnostic method and renders possible the early diagnosis of tuberculosis in all its localizations and forms. Its advantages far outweigh its disadvantages; it is specially valuable in view of its diagnostic certainty. It is applicable to ambulant practice and with sufficient care the results of reactions are harmless. All harmful results can be excluded with certainty if the cases are properly selected, indications and contraindications observed and a scrupulous technique employed.

Rivière says:

> Dangerous reactions in the hands of experts and where contraindications and directions for administrations are faithfully observed, are rare, and do not suffice to condemn the method when applied with adequate skill.

I know of no other certain method of diagnosing the nature of the primary complex or of a tuberculous condition of the lung which is not open. If we are going to wait till all patients have tubercle bacilli in their sputum, we are going to waste much valuable time, make the treatment more difficult and prolonged and diminish the chances of a complete cure.

In the treatment of patients of Type I. start off with 1% tuberculin ointment. This contains: Koch's old tuberculin, 0·5; terebene oil, 10·0; lanoline, 40·0.

Press out a cylinder two centimetres long and rub into the area where there is the lesion, for example over the enlarged bronchial glands.

Notice the skin reaction; there may be a lichen like reaction or there may be blisters. As soon as the skin reacts, stop the treatment till it heals. Watch the temperature. If it is raised, wait two or three weeks till it returns to normal. If a high temperature was produced, use a smaller amount or a weaker ointment.

Each tube of ointment contains twenty grammes. After finishing one tube wait a month before starting on the second strength.

It is usually stated that for Type II. the pubertive type of pulmonary tuberculosis or galloping consumption, there is no specific treatment. Certainly anything in the nature of tuberculin treatment would be quite useless. Nor is it possible to benefit these patients with any form of operative interference. I have seen two methods of treatment used with some measure of success. I refer to "Sanocrysin" and Spahlinger's serum. "Sanocrysin" is a combination of gold and sulphur salts with which Professor Moellgaard obtained very good results when he used it on tuberculous calves. The only patients who appeared to derive benefit were two or three suffering from very acute disease. Many patients developed albuminuria which rapidly

cleared up and I do not think it has been decided whether this is due to the toxicity of the drug or to the action of the toxins which are liberated when the bacilli are killed. It did not appear to benefit patients with fibro-caseous disease.

So much has been written about the Spahlinger cure, such prominence has been given to it in the lay press and so many unsuccessful attempts have been made to have this form of treatment subjected to a thorough scientific test that it is difficult to add anything to what is already known about it. During the last few months Spahlinger has been inoculating some calves in Kent with his vaccine. When he considers them immune they and an equal number of controls are to be infected with tubercle bacilli. The whole of this work is under the supervision of an unofficial though representative committee and their report should be available in the near future. While at Geneva, I examined sixty patients which were still or had been under Spahlinger's treatment and I formed the opinion that he is obtaining exceedingly good results. No selection of his patients is made; he treats them regardless of how advanced their disease may be. The majority of the patients who go to him, have given other methods an extended trial. None of his patients receives the benefit of artificial pneumothorax or of thoracoplastic treatment and on the whole they live under very unideal conditions. That he does obtain good results speaks for the value of the treatment.

Patients of Type III., the proliferating primary complex, are very suitable for subcutaneous tuberculin injections. The tuberculin should be given in exceedingly small doses and the increase made very gradually. These patients are extremely sensitive to tuberculin and it is those who are the most sensitive who eventually give the best results. But in Switzerland, in England and here in Australia it is frequently stated that if a patient react to small doses of tuberculin, he is not suitable for tuberculin treatment.

Strengths of tuberculin solutions are as follows: Strength I. is a one in ten solution (one part old tuberculin and nine parts of 0·25% solution of carbolic acid). Strength II is a one in a hundred solution; Strength III. a one in a thousand solution; Strength IV., one in ten thousand; Strength V., one in a hundred thousand; Strength VI., one in a million and so on. There is no upper limit.

A convenient form of syringe for these injections is one in which the cubic centimetre is divided into twenty parts. Usually the dose can be increased by 50% in the following manner: $\frac{3}{20}$, $\frac{4}{20}$, $\frac{6}{20}$, $\frac{9}{20}$, $\frac{14}{20}$, $\frac{20}{20}$ of a cubic centimetre at intervals of four or five days.

The rules for tuberculin treatment are as follows: We shall assume that the treatment commence with 3/VI. = 0·00015 milligramme of old tuberculin, that is three-twentieths of a cubic centimetre of Solution VI.

If no reaction—febrile, general, focal or local—occurs, then you may give 4/VI. after four days.

If a slight reaction occurs, that is if the temperature is raised 0·1° C. to 0·4° C., repeat the same dose, do not increase it. If again the same reaction occurs, repeat the dose again. It is important to regard every increase in temperature as a reaction. No attempt should be made to explain it in any other way.

If there is a larger reaction such as 0·5° C. to 0·9° C. wait till the fever has quite gone and then give a smaller dose, for example 20/VII. This is a typical reaction but it is too big.

If there is a high reaction, for example 1° C. or more, the dose must be divided by ten, that is, give 3/VII.

If there is a lasting reaction, in other words if for some days the temperature shows an increase then the dose is too high. Give no more injections till the temperature is normal and then give 3/VII. or 3/VIII. Tuberculin should not be given to patients who are febrile, except when there is an effusion into a joint, into a pleural sac or into the peritoneal cavity. Patients in whom the effusion is due to tuberculosis, do very well with tuberculin treatment. The effusion is rapidly absorbed. In these patients who are running a temperature when the tuberculin is given, a sixth reaction might be noted.

Relapse of temperature shows that the dose is effective so do not increase it. Repeat the same dose on the fourth day.

The last point to watch is the amount of urine excreted. In all cases of tuberculosis with effusion, be it into a joint, into the pleura, the pericardium or the peritoneum, the amount of urine should be measured every day. If the effusion be due to the tubercle bacillus, a polyuria should be produced by the tuberculin treatment. This polyuric reaction is very important and many consider it extremely useful in diagnosis, for it has been stated that if there be no polyuric reaction after the injection of tuberculin, then the tubercle bacillus can be excluded as the cause of the effusion.

A reaction which is often seen in old people and in those whose heart muscle is damaged perhaps by the toxins of the tubercle bacillus, is a reduction in the amount of urine excreted. In these patients who give an oliguric reaction, tuberculin treatment must be abandoned, as it is dangerous.

The two members of this type which may go on to ulceration with cavity formation, namely *tuberculosis fibrosa diffusa* and *densa*, usually have small cavities which are bilateral and deeply embedded in dense fibrous tissue. Some of them do very well with tuberculin treatment. This is the type of case which Spahlinger treats with vaccines, often with success.

Type IV. comprises patients who are not suitable for tuberculin treatment, as they are not allergic. Large doses can be given, but they do not produce good results. Members of this group have large cavities often with high temperature and much sputum. Some of the patients are very cachectic, but many appear to be in good condition, being well nourished and able to get about. Often they do not know that they have tuberculosis and are a source

of danger to themselves and to the general public. The dangers to themselves are that they will contract tuberculosis of the larynx or of the intestine or after some exertion, such as a dance or a game of tennis, they will aspirate some of the infected sputum into an unaffected part of their lung and later on become affected by a tuberculous aspiration pneumonia. As soon as the presence of a cavity has been diagnosed, an attempt must be made to collapse its walls. In every case an attempt should be made to produce an artificial pneumothorax. This is the only certain way of ascertaining whether there is a free pleural space or not. Neither the physical examination nor the radiographer's report can be relied on for this information.

Artificial Pneumothorax.

In their "Review of Six Hundred Cases of Pulmonary Tuberculosis Treated by Means of an Artificial Pneumothorax" the Matsons and Bisaillon divide their patients into eight groups and the groups include the following conditions:

Group I., fibro-caseous tuberculosis of a progressive character in which little or no excavation is demonstrable.

Group II., fibro-caseous cavernous tuberculosis.

Group III., fibro-caseous cavernous tuberculosis with cavity manifestations in the foreground.

Group IV., severe acute infiltrating types of tuberculosis, caseous pneumonic and caseous bronchopneumonic forms.

Group V., tuberculous pleuritis with effusion in which there is undoubted evidence of tuberculous involvement of the underlying lung parenchyma.

Group VI., severe and uncontrollable hæmoptysis.

Group VII., bilateral fibro-caseous cavernous tuberculosis.

Group VIII., desolate and hopeless cases in which pneumothorax is produced with the object of controlling severe symptoms and of prolonging life.

From consideration of this classification it will be seen that the main ground for instituting artificial pneumothorax is the presence of a cavity. Progressive caseous conditions and uncontrollable hæmoptysis are also included. Professor William Neumann says that the main indications are the presence of a cavity and uncontrollable hæmoptysis.

Early unilateral disease is regarded by some as an indication for this form of treatment, but it is fairly generally agreed that patients in the early stages should be given the benefit of sanatorium treatment first and, if after six months it is found that the disease is still progressing either in extent or in character by going on to caseation and liability to breakdown with the formation of cavities, then artificial pneumothorax should be produced.

The condition of the contralateral lung must receive very careful consideration. Many pathologists state that in a patient with disease sufficiently far advanced in one lung to require this form of treatment the contralateral lung will always be found more or less diseased. So long as not more than one-third of the better lung is involved or so long as active disease does not extend below the level of the third rib in front and the spine of the scapula behind, the outlook is fairly hopeful. When the process in the contralateral lung is very active, it is better to wait for this activity to subside somewhat before instituting pneumothorax treatment. The danger is that during this period of waiting adhesions might form, thus rendering the production of an artificial pneumothorax impossible. The Matsons have eliminated this danger in many of their cases by introducing only sufficient gas to separate the lung from the chest wall and maintaining this very slight degree of compression, in the meantime observing the behaviour of the opposite lung. On numerous occasions they have noted the beneficial influence of a minor degree of compression of the worse side and have been agreeably surprised to observe the retrogression of activity in the contralateral lung. Throughout the whole treatment a most careful watch must be kept on the condition of the other lung. It is necessary to chart at frequent intervals the results of physical examination and to take and compare X ray photographs. If during the pneumothorax treatment there be an unexplained amount of sputum containing bacilli or an unexplained fever, then it is necessary to decide whether the compressed lung is at fault or if the disease is progressing in the contralateral lung. If the former is the case more compression is required, if the latter, further compression is contraindicated.

Burrell in his report on the value of treatment of pulmonary disease by artificial pneumothorax gives the following contraindications:

(i.) Disease of one-third or more of the better lung.

(ii.) Extensive tuberculous disease elsewhere, such as enteritis. Tuberculous laryngitis is not a contraindication and is usually improved by the treatment.

(iii.) The existence of pulmonary tuberculosis as a terminal infection or as an intercurrent infection with some other disease such as diabetes, cirrhosis of the liver, chronic nephritis *et cetera*.

(iv.) A highly neurotic temperament. Patients with a neurotic temperament do badly.

(v.) Ill nourished patients. Patients with chronic dyspepsia or severe visceroptosis.

(vi.) Asthma and emphysema are not always absolute contraindications, but pneumothorax should only be done on such patients if there are special or urgent indications.

(vii.) Tuberculosis in an early stage. Patients with good resistance in the early stages of pulmonary tuberculosis should first be treated with ordinary medical methods.

Two or three complications of artificial pneumothorax treatment must be mentioned:

(i.) Subcutaneous emphysema may be very serious when it involves the mediastinum and may require a tracheotomy.

(ii.) Pleural shock and air embolism. On reading through the literature I find it difficult to separate the symptoms produced by pleural shock from those produced by embolism.

Pleural shock might develope when the pleura is being anæsthetized as in the case described by Stivelman. Some of the patients complain of sudden difficulty in breathing and of faintness. Neumann says that what has been described as pleural shock is really due to gas embolism and the Matsons state that there is no uniformity of symptoms by which they can differentiate sharply between pleural shock and gas embolism at the time of their occurrence.

Air embolism may show itself by the development of a hemiplegia or of a monoplegia, by spastic contractures, by twitching of the face, by aphasia or by sudden blindness. Some patients turn round their own axis or become quite demented, laugh uncontrollably and do not recognize their physicians. The symptoms depend on the position of the gas bubbles, if they are in the internal capsule hemiplegia is likely to result, while bubbles situated deeper in the *corpus striatum* are likely to produce contractures or clonic spasms. The most frequent first sign is a little twitching of the face or movement of an arm. So it is necessary to instruct the patient to keep perfectly still while the needle is being introduced and the operator should keep a careful watch on the patient's face. If this is seen to twitch at all or the arm to move, a movement which the patient says he cannot control, then the needle must be withdrawn at once. When making the fillings it is a good plan to have the head the lowest point and the legs raised on a cushion. Then the patient may complain of a parasthesia or red spots may be seen to develope underneath the skin due to capillary emboli. If the needle be inserted and there is not a good negative vibration the needle is not in the free pleural space. It is then necessary to insert it at another point, but this should not be done on the same day. It is safer to wait till the next day. By doing this and by using a needle of large bore and with a blunt edge the liability to gas embolism will be reduced to a minimum. It is a safe rule never to allow the gas to enter till there is a good negative movement on the manometer and to be guided in the size of the fillings by the manometer readings and by the feelings of the patient. If there is something in the neighbourhood of a negative pressure of -8 in inspiration and -3 with expiration it is absolutely certain that the needle is in the pleural space.

If during the induction of local anæsthesia with "Novocaine" the patient complains of a bitter taste in his mouth, the needle has entered a pleural adhesion and this is good evidence that the point chosen is not a suitable one at which to enter the pneumothorax needle. If during inspiration there is a negative pressure of say -5 and the same negative pressure during expiration there is probably liquid in the needle. A negative pressure of -2 on inspiration and a pressure of $+2$ on expiration suggests that the needle has passed through lung tissue into an alveolus. It would be dangerous to allow the gas to flow as it might produce embolism of the brain. If there is no vibration of the manometer fluid the needle is either in the skin or in a dense pleural adhesion.

A third complication of artificial pneumothorax is pleural effusion. A serous exudate forms in about 20% of cases and is not important so long as the amount is small. The incidence of this effusion is diminished by giving large fillings at long intervals, for example three or four weeks instead of small fillings every three days. Whenever Neumann uses artificial pneumothorax treatment, he always combines it with tuberculin injections. By this means he claims to get only 10% of his patients with pleural effusions. An effusion usually follows the separation of an adhesion and causes a rise in temperature. If it be allowed to remain, it is said to produce a thickening of the pleura and this would delay or prevent the reexpansion of the lung when the treatment is stopped.

A purulent effusion results from one of three causes, the tearing of a piece of lung tissue in an adhesion, the rupture of a superficial caseous focus or a spontaneous pneumothorax. It is important to aspirate early and often and it may be necessary to wash out the pleural sac with saline solution or a weak antiseptic lotion. In only a few cases it is necessary to insert a small drainage tube. Often it is not possible to continue the pneumothorax treatment owing to the rapid obliteration of the pleural space.

The other indication for the production of an artificial pneumothorax is uncontrollable hæmoptysis. The treatment of hæmoptysis should start off with the administration of a sedative mixture such as:

Sodium bromide	10·0 grammes
Sodium chloride	20·0 grammes
Water	200·0 mils.

Give ninety cubic centimetres as soon as the bleeding starts and continue with thirty cubic centimetres three times a day until all sign of hæmorrhage has stopped. It is a mistake to administer morphine or any derivative of opium except in very minute doses. These drugs certainly quieten the patient, but they diminish the cough reflex also and then there is a danger that some of the infected blood and sputum will be aspirated to other parts of the lung and a tuberculous aspiration broncho-pneumonia result. In cases of very severe hæmorrhage it is useful to inject the following solution intravenously:

Sodium chloride	1·0 gramme
Water	10·0 mils.

If these methods fail an artificial pneumothorax should be produced. It is not always easy to decide which lung is bleeding but profuse hæmorrhages are nearly always produced by the ulceration of a vessel in a cavity.

When three or four unsuccessful attempts have been made to produce an artificial pneumothorax, it

is necessary to consider other forms of operative interference such as phrenicotomy, fat or paraffin filling and thoracoplasty. It has recently been shown that in the operation of phrenicotomy it is not sufficient to divide the phrenic nerve in the neck. To produce paralysis of the diaphragm it is necessary to evulse the distal part of the nerve. It was found that after the division of the nerve the corresponding half of the diaphragm was not always paralysed. The cause was found to be that the phrenic nerve is often joined by a twig from the nerve to the subclavius muscle and this was able to carry impulses down to the diaphragm and thus keep it functioning. When the nerve is evulsed this twig is torn across. The result of this operation is that the diaphragm ascends into the chest and the lower part of the lung moves very little with respiration and is rested. It is particularly valuable when pleural adhesions over the lower part of the lung interfere with an otherwise satisfactory artificial pneumothorax. Phrenicotomy is also worth trying for an early one-sided tuberculosis which has not responded to sanatorium treatment. It tends to favour the healing of the lung and to diminish the amount of sputum.

By means of the operating thoroscope it is possible to divide some pleural adhesions but it is an operation that is not without danger. When through inability to find a free pleural space it is impossible to produce an artificial pneumothorax, an attempt should be made to collapse the lung by means of thoracoplasty. In the hands of experienced surgeons this operation gives brilliant results. Brauer, Wilms and Sauerbruch in Germany and Switzerland, Denk in Vienna, Alexander in Michigan and Morriston Davies in Wales are obtaining excellent results. For this operation the patients must be most carefully selected and the condition of the contralateral lung calls for just as careful examination as it does when artificial pneumothorax therapy is under consideration. The operation is performed in two stages. In the first stage five centimetres of the eleventh to the sixth ribs are resected and two or three weeks later a similar amount is removed from the remaining five ribs. It is often not possible to remove more than three centimetres of the first rib. On the Continent the operation is performed under local anæsthesia, but in America they use a combination of local and "gas" anæsthesia. Sometimes the patient has not sufficiently recovered from the first stage to allow the operation to be completed in three weeks. If there is a longer interval than two months a bony bridge forms between the ends of the ribs and this has to be removed when the second stage is being done. Within forty-eight hours there is a rise of temperature which soon settles down, the quantity of sputum is diminished and the patient rapidly feels improved. Remarkably little deformity is produced by this operation and scoliosis and displacement of the heart, produced by the fibrosis and its consequent breathlessness, are relieved by thoracoplasty. At the end of 1925 Professor Denk gave me the following figures of his results in seventy-six cases: Proliferative type of tuberculosis, 5% deaths; exudative type of tuberculosis, 24% deaths. Over the whole series of seventy-six cases his mortality was 14%. When it is remembered that all these patients were in an advanced stage of the disease, it must be admitted that the results are very good. These results correspond very closely to those of Sauerbruch, but it is only these very experienced surgeons who can show such a low mortality rate. Almost more important than the experience of the surgeon is the selection of the patients whom the physician considers suitable for the operation.

A fourth procedure for the collapse of cavities is extrapleural pneumolysis in which fat or paraffin is introduced. This operation is often combined with phrenicotomy.

Other Types of Specific Treatment.

Some other types of specific treatment I must refer to quite briefly. Dreyer's defatted vaccine has received an adverse report from a committee appointed by the Medical Research Council. A similar committee issued in *The Lancet* of November 3, 1923, its report on Camac Wilkinson's tuberculin treatment. I shall quote one paragraph:

> There is no doubt that this method of tuberculin administration succeeds in immunising patients to large doses of tuberculin, but there is no evidence that it also immunises them to tuberculosis.

When discussing the therapeutic use of tuberculin, I suggested that it was those patients who were very allergic to tuberculin who did best with tuberculin treatment. Many specialists on the Continent consider that patients who stand large doses of tuberculin, are not suitable for this class of treatment.

Heliotherapy.

While all are agreed that heliotherapy and ultraviolet light are valuable in the treatment of all kinds of surgical tuberculosis, there is still much doubt about its use in pulmonary tuberculosis. While visiting Leysin at the end of 1924, I was very impressed with the fact that while there were no blinds at the clinics controlled by Dr. Rollier, all the sun boxes of the sanatoria higher up were provided with blinds or screens. These sanatoria are devoted to the treatment of patients with pulmonary conditions, while Dr. Rollier does not admit such patients to his clinics. I gathered that it is the general opinion that heliotherapy is not suitable for these pulmonary conditions. With this view Dr. Rollier does not agree, for he says that many of his surgical patients suffer from conditions complicated by pulmonary lesions and he has not seen any of them made worse by his sun cure. But with these patients he increases the dose very gradually and is perhaps more on the alert for any sign of reaction. Ultraviolet light treatment by means of the mercury vapour lamp or by the carbon arc is considered bad for pulmonary tuberculosis. Dr. Sequiera who is in charge of the Light Department of the London Hospital has seen conditions caused to flare up by its use.

Sanatorium and General Treatment.

I have made no mention of the value of rest and of ordinary sanatorium treatment and have endeavoured to confine my remarks to specific methods of treatment. Rest, fresh air, sunlight and good food are always essential, rest being the most important.

Summary.

In tuberculosis of Type I., simple primary complex with glandular involvement tuberculin and perhaps heliotherapy should be used.

In tuberculosis of Type II., the rapidly progressive, pubertive type of pulmonary tuberculosis, no form of operative or active immunizing treatment is possible. Passive immunity by means of Spahlinger's serum or treatment with "Sanocrysin" is recommended.

In tuberculosis of Type III., proliferating primary complex, try to produce an active immunity by subcutaneous tuberculin or by Spahlinger's vaccines.

In tuberculosis of Type IV., cavities require operative treatment in the form of artificial pneumothorax, phrenicotomy, extrapleural pneumolysis or by thoracoplasty.

THE SURGICAL ASPECTS OF TUBERCULOSIS.[1]

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THE subject of the surgical aspects of tuberculosis is so vast that it is obvious the dictates of time permit only a reference to some of them. It is the surgeon who more than anyone sees the ravages wrought by the surgical forms of tuberculosis and who knows the patchwork nature of the cures so laboriously achieved, so that it is natural that he should be keenly interested in the evolution of methods of preventing tuberculous infection. It is possible that I may repeat something of what the speaker on the preventive aspects of tuberculosis has said. If so, repetition will but serve to impress on all the compelling importance of prevention.

Koch's identification of the tubercle bacillus with the disease and much later his famous pronouncement before the Tuberculosis Congress held in London at the end of the last century, on the rôle played by the bovine bacillus in tuberculous infection, have not had the effect that was expected in eradicating or even in diminishing the disease. Far more effort and money have been expended in the detection and treatment of the disease than in its prevention. In opening the Congress referred to, the late King Edward the Seventh, then Prince of Wales, asked, *à propos* of tuberculosis, the now historic question, "If preventible, why not prevent it?" The same question may be asked with as much insistence today. The human and bovine carriers of the disease, in full view of an enlightened generation, still stalk the land and disseminate the disease. The attainment of a pure milk supply and the production in the human individual of a lifelong resistance to tuberculous infection are the ideals and essentials of a successful prophylaxis.

Stanley Griffiths in the investigation of a large number of cases of surgical tuberculosis occurring in children under ten years of age recorded the results in the accompanying Table I.:

TABLE I.—SHOWING CASES OF SURGICAL TUBERCULOSIS IN CHILDREN UNDER TEN YEARS OF AGE.

Condition.	Number of Cases.	Human.	Bovine.	Percentage Bovine.
Cervical gland tuberculosis	47	14	33	70·2
Bones and joints (England)	288	217	71	24·6
Bones and joints (Scotland)	22	14	8	36·3
Lupus	89	40	49	55
Total	446	285	161	36

Thus the incidence of bovine tuberculosis in children under the age of ten is about 36%. Table II. gives the results of a parallel investigation in children over the age of ten:

TABLE II.—SHOWING CASES OF SURGICAL TUBERCULOSIS IN CHILDREN OVER TEN YEARS OF AGE.

Condition.	Number of Cases.	Human.	Bovine.	Percentage Bovine.
Cervical gland tuberculosis	69	48	21	30·4
Bones and joints (England)	188	172	16	8·5
Bones and joints (Scotland)	6	6	—	—
Lupus	37	22	15	40·8
Total	300	248	52	17·3

From these tables it may be concluded, as Frazer in his "Surgery of Childhood" writes, that the bovine bacillus "is a special menace to the child."

He also says:

> The problem of the tuberculous cow is an economic one. The ideal would be the destruction of the animal as soon as it was demonstrated that it was the victim of tuberculosis.

It may be granted that wholesale destruction of the tuberculous cow would be an expensive procedure, but the many and various measures taken for the detection, care and treatment of human beings suffering from tuberculosis, also entail great expense. As the eradication of tuberculosis from its midst would benefit the whole population of the Commonwealth, I suggest that the destruction of every tuberculous cow and its replacement by a healthy high grade milker at the expense of the State, is an economic possibility. The breeding of

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 29, 1926.

healthy herds of high grade milkers in State farms in each State would have a twofold effect. It would go a long way towards stamping out bovine tuberculosis and it would also raise the standard of dairy cattle to the great benefit of the dairying industry. I maintain it would be a better financial proposition for the State to breed and dispense healthy cows, than to maintain tuberculous human beings.

The production of resistance in the human subject to tuberculosis, in other words the artificial immunization against tuberculous infection, is a problem less easy of solution. But recent researches by Calmette are encouraging in the highest degree. He has long applied himself to the possibility of protective vaccination of susceptible individuals. The vaccinating strain he used, was originally a very virulent bovine culture which since 1908 has been continuously grown on potato cooked in ox-bile with 5% glycerine. After thirteen years and some two hundred and thirty subcultures, this strain was found to have lost virulence for all species of animals, but to be antigenic in the sense that inoculation excites the formation of antibodies demonstrable by complement fixation. Three doses of one centigramme of the living culture were swallowed in a little milk half an hour before a feed on the fourth, sixth and eighth days or the fifth, seventh and ninth days after birth. In June, 1922, 178 nurselings were vaccinated and kept under subsequent observation. Up to June, 1925, fifteen had died and in no instance was death from tuberculosis. This death rate of 8·4 compares with one of 11·6 for infants of the same age in the whole of France in 1921. Since July, 1924, 1,070 new-born infants have been vaccinated in France and Belgium. Precise information in July, 1925, was obtainable of 423 who had been vaccinated for six months or longer. One hundred and thirty-seven had been exposed to tuberculous infection in the family; of these eighty-six had a tuberculous mother. The deaths numbered thirty (7%) and there was no death from tuberculosis. As a control, 1,362 tuberculous mothers who in 1922 had 1,364 children, were taken. Of these 327 (24%) died before July, 1925. Statistics were also obtained from various dispensaries. In short an infant, born of a tuberculous mother and not separated from the mother, has one chance in four, or in certain places, such as Paris, three chances in four of dying of tuberculosis in the course of the first year. Against this is placed one death from tuberculosis among 137 infants who lived more or less constantly exposed, since birth, to family infection.

These results of Calmette may inspire us with hope for the future. In the meantime more stringent measures should be taken to prevent close and prolonged contact of others with a tuberculous individual. Children cannot protect themselves and therefore they should be protected, particularly in the early years of childhood, when they are most susceptible to infection. There is no reason why in the future a tuberculosis resisting human race should not be evolved just as a rust resisting wheat is a reality of the present. The diagnosis of surgical tuberculosis is at times shrouded in difficulty. Is the discovery of the tubercle bacillus to be the only positive test of a pathological condition being tuberculous? In cases of "closed" tuberculosis it is impossible to insist on this. Failing the discovery of the tubercle bacillus are the microscopical features of a section of diseased tissue conclusive evidence of a tuberculous condition? Caution is necessary as the following case shows:

A middle aged woman with an ulcerating lump in the upper quadrant of the right breast, was sent to me for the purpose of having her breast removed for tuberculosis. Inquiry revealed that the diagnosis had been based on the histological appearances of a section and not on the discovery of the tubercle bacillus. The patient did not look tuberculous, no other tuberculous foci could be discovered and the clinical signs suggested syphilis rather than tuberculosis. The ulcer healed and the infiltration of the breast rapidly disappeared under antisyphilitic treatment.

While the presence of a tubercle bacillus is conclusive evidence of the tuberculous nature of a lesion, its absence does not prove the contrary.

I have for example met with two cases of slight symptomless hæmaturia; one patient was the subject of tuberculous disease of the knee joint, the other had tuberculous disease of the right shoulder joint, the spine, the right hip joint and both testes. In neither case could the tubercle bacillus be found in the urine on repeated search, nor was inoculation of a guinea pig any more successful.

The hæmaturia in these cases must, I think, be due to a "closed" tuberculous focus in the kidney.

Prior to the discovery of the X rays and their skilful use in diseases and injuries of the hip joint in the young, it is certain that many conditions diagnosed as tuberculous were not examples of that infection. Recovery in these cases was regarded as being due to the success of the treatment adopted in the belief that the condition was tuberculous. We know now that many of such patients were suffering from Perthes's disease. The mistake is, I am sure, still made. To clear up doubts when they exist, it is essential that the radiograph be of the finest definition. A poor radiograph tends only to perpetuate the doubts.

A year or so ago I treated a fat, overgrown boy for several months for what I believed to be tuberculous disease of the left hip joint. An indifferent radiograph seemed to confirm the diagnosis. A few months ago the lad had an epileptic fit in his sleep and afterwards had similar clinical signs in the other hip. A radiograph showed that he had displaced the upper epiphysis of the right femur. It was then realized that the old affection of the left hip was probably of a similar nature and inquiry elicited that the symptoms had been caused in a similar way. A good radiograph showed the epiphyseal displacement to be bilateral.

Certain conditions of the vertebræ, produced by trauma or by an infection, may closely simulate tuberculous disease of the spine.

Two years ago I was asked to see a doctor's son, aged sixteen, who a day or so after practising the high jump for his school sports complained of pain in the dorsi-lumbar region of the spine which radiated to the abdomen chiefly in the right iliac region. There was muscular rigidity in the affected region of the spine and the pain caused loss of sleep. As there was a family history of tuberculosis, Pott's disease was suspected. After a few weeks' rest the symptoms abated and his father decided to take the lad

for a sea trip. On his arrival at Melbourne the symptoms were as prominent as ever. A leading surgeon was consulted and a distinguished radiologist confirmed his diagnosis of tuberculous disease of the spine. The patient returned to Adelaide with a dubious father. However the usual treatment of rest, open air, sunlight *et cetera* was adopted and continued for three months. At the end of that time all pain had gone and his still dubious father allowed him to get about again in a natural way. He has remained well ever since and radiographs reveal no sign of disease. I have little doubt that the symptoms in this case were due to trauma of a vertebral epiphysis or of an intervertebral disc.

A spinal affection which mimicked Pott's disease was met with in a doctor, aged about fifty-two. Following an attack of influenza he quite suddenly was seized with severe pain in the mid-dorsal region which radiated to the epigastrium. The diagnosis of an affection of the spine was confirmed by a radiograph. The radiographic films showed very definite absorption of the front of the adjoining portions of the bodies of the sixth and seventh dorsal vertebræ. A consultant thought the appearances taken in conjunction with the severe root pains were in favour of sarcoma. I advised treatment by rest in the belief that the condition was tuberculous. The treatment was carried out in a perfunctory manner for a few months and the patient then returned to a large general practice and drove his motor car over bad suburban roads.

Some will doubtless say that the two cases which I have related were examples of tuberculosis occurring in individuals whose resistance was high. I believe, however, that Pott's disease, like hip disease, may be simulated by other infective conditions which bring about a rarefying osteitis. Caution is necessary in making a diagnosis which in most cases entails prolonged treatment for three years.

The operation wounds made for the treatment of surgical tuberculosis as a rule heal very readily, but occasionally the low resistance to tuberculosis is accompanied by a depressed state of the unaffected tissues so that an incision made through them refuses to heal.

I have in mind an aboriginal on whom I had performed an extensive resection of the bowel for ileo-caecal tuberculosis. A muscle splitting incision was employed and apparently healed soundly. Three weeks after the operation the wound broke down throughout and the bowel prolapsed. Secondary section was performed without effect. The patient died from miliary tuberculosis.

I have seen an incision made for the removal of a tuberculous kidney and ureter behave in a similar way.

A massive soiling of the wounds with tuberculous tissue would afford another explanation of the failure to heal.

In other cases the removal of the incubus of several coincident tuberculous infections has a most pronounced effect on the general health.

I recall the case of a girl, aged sixteen, whose foot I amputated for an almost fulminating tuberculous disease of the ankle joint and whose knee and elbow joints on the same side required excision. She is now fat, rosy and well.

The treatment of surgical tuberculosis is rightly becoming more and more conservative, but there are three conditions in which operative measures are to be preferred—tuberculous lymphadenitis, tuberculous peritonitis with ascites and tuberculous disease of the knee joint in adolescents and adults. A well performed excision of the knee joint will enable a manual labourer to return to work with a strong and useful limb in six months. Few operations in surgery give a more gratifying result.

The rarity of lupus and tuberculous dactylitis in this land of sunshine is well known and is a strong argument in favour of the employment of sunlight in the treatment of the disease. To get the best results sunlight must be employed intelligently and in no haphazard manner. There is great scope in this country for research on the subject. It would probably lead to valuable information on the therapeutic uses of sunlight.

THE MANIC-DEPRESSIVE PSYCHOSIS.

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Dr. Lind's article in The Medical Journal of Australia of July 17, 1926, on the symptom-complex of recurrent mental states, known as manic-depressive insanity, amounted more to a cavilling at technical terms and a plea for the rehabilitation of the Cloustonian nomenclature than to any very trenchant or elucidating criticism of the mental malady.

Obviously there exists some confusion of terms and in the lack of more positive knowledge for which we naturally look to pathologists and bio-chemists, this is bound to be. But mania and melancholia, as I understand them, are symptoms merely and not separate morbid entities. They are psycho-clinical manifestations of a very definite symptom-complex which modern psychiatrists, following Kraepelin, call the manic-depressive psychosis. Many other similar symptoms, such as the motor restlessness and excitement seen in certain cases of *dementia præcox*, acute confusional insanity (often wrongly termed acute delirious mania) and the furor of epilepsy, are frequently confused with the mania of manic-depressive insanity. Likewise many states of depression, including involutional melancholia and even senile dementia and catatonia, are confused with the depression of manic-depressive insanity. But to my mind manic-depressive insanity is a clear and definite syndrome.

Naturally as a symptom-complex manic-depressive insanity may and does exist in one of a number of forms. Those of easy recognition clinically are: Repeated attacks of mania with sane intervals (called recurrent mania); repeated attacks of depression with sane intervals (called recurrent melancholia); alternate attacks of exaltation and depression with a sane interval (called alternating insanity); repeated attacks of mania followed by depression, the one merging into the other without any appreciable sane interval (called *folie circulaire*); repeated attacks of mania followed by depression with a definite sane interval following each cyclic attack.

Examples of these forms of manic-depressive insanity I have seen repeatedly and I find that my experience very largely corresponds with that of Dr. George M. Robertson[1] who succeeded Clouston at the Royal Infirmary at Morningside (Scotland).

The following is an analysis of the condition of thirty-two male patients suffering from manic-depressive insanity at present in Sunbury. The age in each instance is that of the patient at the time of his first attack and the description is that of the number and type of subsequent attacks:

Aged 35, four attacks of acute mania.
Aged 27, repeated attacks of excitement and exaltation.
Aged 31, *folie circulaire*.
Aged 40, alternating insanity. Has an insane heredity.
Aged 19, one attack of acute mania.
Aged 20, repeated attacks of mania.
Aged 27, one attack of acute mania.
Aged 45, recurrent attacks of depression.
Aged 17, unstable temperament; repeated alternating attacks.
Aged 15, alternating insanity.
Aged 18, alternating attacks of mania and depression for past thirty years.
Aged 41, alternating insanity.
Aged 41, repeated attacks of mild mania.
Aged 24, two attacks of mania; is at present normal.
? Repeated attacks of mania with short remissions since 1903.
Aged 50, repeated attacks of acute mania.
Aged 35, first attack of mania seventeen years ago; now recurrent.
Aged 42, *folie circulaire*—manic phase predominates.
Aged 32, two attacks of depression.
Aged 23, two attacks of depression; is now well.
Aged 43, repeated attacks of depression.
Aged 30, repeated attacks of mania.
Aged 18, repeated attacks of depression with good remissions.
Aged 31, *folie circulaire*.
Aged 31, two attacks of mania; is now well.
? mixed insanity. Irregular attacks of mania and depression.
Aged 32, two attacks of mania.
Aged 38, four attacks of mania; has good remissions.
Aged 32, recurrent mania.
Aged 23, *folie circulaire* with the depressive phase predominant.
Aged 25, repeated attacks of exaltation.
Aged 49, *folie circulaire*.

The following is an analysis of the condition of fifty-eight female patients suffering from manic-depressive insanity at present in Sunbury. The conditions were variously diagnosed as confusional, non-systematized insanity, chronic mania, recurrent melancholia and general paralysis of the insane. The age in each instance is that of the patient at the time of the first attack and the description is that of the number and type of subsequent attacks:

Aged 36, more than six attacks recorded.
Aged 21, two attacks of mania.
Aged 45, *folie circulaire*, many attacks.
Aged 14, four attacks of mania.
Aged 36, five attacks of depression.
Aged 32, more than four attacks of mania.
Aged 40, moral insanity, alternately depressed and exalted.
Aged 38, attacks of mania (has goitre).
Aged 34, four attacks of mania.
Aged 49, *folie circulaire*.
Aged 31, five attacks—alternating.
Aged 18, alternating depression and mania.
Aged 29, three attacks at long intervals.
Aged 36, repeated attacks of mania.
Aged 44, *folie circulaire*.
Aged 35, *folie circulaire*.
Aged 43, manic phase predominates.
Aged 46, repeated attacks of mania.
Aged 39, five attacks of mania and depression.
Aged 44, acute mania.
Aged 26, *folie circulaire* (many attacks).
Aged 37, six depression, two mania.
Aged 28, three attacks of mania, attempted suicide.
Aged 28, three attacks of mania.
Aged 26, *folie circulaire*.
Aged 34, repeated attacks of mania.
Aged 40, six attacks of depression.
Aged 40, three attacks of mania.
Aged 35, repeated attacks of mania.
Aged 45, acute mania, now recovered.
Aged 31, *folie circulaire*.
Aged 35, true alternating insanity.
Aged 21, *folie circulaire*.
Aged 46, repeated attacks of mania.
Aged 35, *folie circulaire*, with depressive phase predominant.
Aged 27, repeated attacks of depression.
Aged 39, four recorded attacks of mania.
Aged 40, *folie circulaire* with long remissions. She is really a typical case of what some writers call "*folie à double forme*."
Aged 37, repeated attacks of depression.
Aged 27, *folie circulaire*.
Aged 21, repeated attacks of mania.
Aged 17, admitted in an extremely depressed state, now becoming exalted.
Aged 30, four attacks of depression, one of mania.
Aged 43, *folie circulaire*.
Aged 31, recurrent mania.
Unknown, *folie circulaire*.
Aged 20, recurrent mania.
Aged 36, alternating insanity.
Aged 40, *folie circulaire*.
Aged 31, *folie circulaire*, with depressive phase predominant.
Aged 19, *folie circulaire*; has also been in three asylums in Scotland.
Aged 17, recurrent attacks of depression.
Aged 19, *folie circulaire*.
Aged 18, alternating insanity, alternations occur almost monthly.
Aged 27, *folie circulaire*.
? *folie circulaire*, with manic phase predominant.
Aged 30, four attacks of mania.
Aged 39, recurring attacks of mania.

It should be noted that in order to save confusion all cases of involutional melancholia and senile agitated melancholia have been rigidly excluded from this analysis.

In the analysis of male patients it was found that a number of conditions had been wrongly diagnosed as confusional, adolescent insanity, dementia and two patients were thought to be suffering from general paralysis of the insane.

The average age of onset for males is thirty-one and that for females thirty-two years.

An insane heredity was recorded against 33% of these patients in the clinical history sheets.

In reference to Dr. Lind's article, I fail to see why "in the question concerning the appropriateness of the term manic-depressive insanity, the evidence for and against is not complete till the death of the patient." I fail to see why in the undoubted and acknowledged absence of specific *post mortem* findings which might be used to confirm a diagnosis, it matters in the least if a patient dies during a period of mania or depression or recovering from

either of these, is discharged from hospital to die perhaps in comparative sanity. It does not matter one iota whether a patient has two attacks of mania and dies or has twenty-two attacks and recovers. The whole point is that if at any time during life a patient manifests the symptoms of manic-depressive insanity, the condition should be diagnosed as such. So that Dr. Lind's *"post mortem* statistics" have no superior claim over any other observant psychiatrist's "vital statistics."

In fact, statistics are very often misleading. It is related of an eminent statesman that when he wished to confound his opponents he would do so with statistics. And it would appear that Dr. Lind, having let off a few squibs at Craig and Stoddart, has opened fire on Kraepelin under a statistical smoke-screen. I regret that his statistics are unconvincing, perhaps because, working each day among the case records of the Victorian Lunacy Department, I am only too conscious of their many and gross inaccuracies. And I am loth to place any reliance upon the "personal statement" of patients accompanying the medical certificates in which questions concerning previous attacks and habits of life are in nine cases out of ten filled in by some illiterate aunt or the local constable. Take Dr. Lind's first set of figures. We are told that one hundred and sixty-two patients had "one attack only," sixty-five patients had two attacks, nine had three attacks and so on. What were they attacks of? Excitement, confusion or lucidity? Has not the fly of fallacy already crept into the statistical ointment? And what is "recurrent insanity other than mania and melancholia" (in the same set)? If one is to regard the remissions of a general paretic, the frequent outbursts of a dipsomaniac or the several possible attacks of puerperal insanity as "recurrent insanity," what have they to do with the manic-depressive psychosis under discussion? And why drag in fourteen vague cases of "not first attack"? Such nebulous information sheds no statistical light whatever. Again, in another group under the heading of "three attacks" Dr. Lind discloses the fact that one individual had "two attacks unknown" and a third of "terminal dementia." Now, if these two attacks are unknown, does Dr. Lind assume that they were "manic-depressive"? If not, why are they put into his statistics? Likewise in the next group, if the patient who is said to have had four attacks of "puerperal mania," really did have puerperal insanity, what possible connexion can it have with manic-depressive insanity and of what value is it except to obfuscate further some already misleading figures? I submit, therefore, that these statistics from which conclusions are drawn, are as barren as the historical fig tree at Bethany.

Dr. Lind has grievously misread the valuable writings of Kraepelin, if he imagines that because one hundred and sixty-two of his patients out of two hundred and seventy-five having one attack either of mania or melancholia, showed no "periodicity," therefore they cannot have suffered from manic-depressive insanity. The cognomen at which Dr. Lind so persistently baulks was coined by Kraepelin to embrace and differentiate certain disorders of the affective state "in the realm of volition and action." Periodicity is in no sense a determining factor, for 68% of Kraepelin's patients had only one attack.[2] And Dr. Lind fails in critical acumen in asserting that his statistics show that "there is no justification for the theory that mania and melancholia are phases of a disease called manic-depressive insanity."

I cannot agree with Dr. Lind's derisive parallel wherein he condemns manic-depressive insanity because, forsooth, a person may have "tracheitis, bronchitis, bronchopneumonia, lobar pneumonia or pleurisy without the attacks being called phases of the same disease." Obviously and for the very excellent reason that the diseases above mentioned are due to different pathogenic microorganisms. But if a person becomes affected by tuberculous laryngitis which spreads into his chest, setting up a tuberculous pleurisy and finally a tuberculous bronchopneumonia, then will these be phases of the same disease. But the parallel will not bear intelligent criticism, because the respiratory disorders enumerated by Dr. Lind have been named in respect to their morbid pathological states, while the various phases of manic-depressive psychosis have been named in the only way possible in the present state of our knowledge and that is from the psycho-clinical symptoms which they present.

Kraepelin's conception of manic-depressive insanity is of value in modern psychiatry for two reasons, namely: (i.) That it enables one to differentiate a certain psychosis from a very large group of psychoses, occurring in early adult life and previously lumped together with much confusion under headings of adolescent insanity, juvenile mania, melancholia and *dementia præcox.* (ii.) That it supplies a theory which will at least explain the symptoms which the disease manifests.

McDougall[3] has recently pointed out that manic-depressive insanity may be regarded as a psychogenic disorder and that as the normal man's estimate of himself and his surroundings is based upon the balance struck between the instincts of self-assertion and self-abasement, any grave periodical or single upsetting of this equilibrium will tend to produce the syndrome of manic-depressive insanity. He suggests three ways in which this personality equilibrium may be deranged, namely: (i.) by the influence of external circumstances, calamity *et cetera,* (ii.) by some disorder of body metabolism, either of a toxæmic or endocrinic nature and (iii.) some inherited instability of the sentiment of self-regard, leading to a dissociation of the two instincts. It was on a temperamental basis that Kraepelin postulated his theory of manic-depressive insanity. And, developing his ideas, later investigators have separated temperament into the cyclothymic and schizothymic varieties, normal personality residing in a blending of the two. If the cyclothymic, being in excess, becomes dissociated, some form of the

manic-depressive psychosis will be manifest. If the reverse be the case, some form of *dementia præcox* will result.

Now, the manifestations of psychic life are greatly influenced by the biochemical activities of glandular mechanisms. The late Frederick Mott[4] showed in a series of brilliant pathological studies that *dementia præcox* is due to genetic inadequacy and, arguing from what has been stated above, one is justified in looking in the same direction for the cause of manic-depressive insanity. The late Clifford Allbutt[5] hinted that the secret lay hidden in some ductless gland. And now Dercum[6] has advanced a theory which promises to shed much light upon the matter. He divides the endocrine glands into two groups—catabolic and anabolic. Into the first he puts the pituitary, thyreoid and the chromaffin system and into the second those glands which have an opposing action, namely the thymus, parathyreoid and pancreas. Dercum claims that normally these glands exert a stabilizing control upon the thalamic nuclei; that if there is any hypofunction of the catabolic group, metabolism will be retarded, thought will be slower, emotional pain and depression will result; on the other hand, if there is any hypofunction of the anabolic group the reverse will take place, the speed of impulses will be accelerated across the synapses, metabolism and cerebration will be increased with motor restlessness and exaltation. Theory only as this is, it is not without great attraction to those who are dealing with manic-depressive patients, because all phases of the disease may be explained by it in harmony with Kraepelin's original conception.

It may be that Dr. Lind has merely set up cock-shies for an intellectual exercise; but the term manic-depressive insanity has gained an almost universal acceptance and the time for quibbling is past. We are waiting for another investigator who, like Mott, will follow the lead of Kraepelin and give pathological proof to the veracity of theory.

Acknowledgment.

I desire to thank Dr. J. K. Adey, Medical Superintendent of Sunbury, for his permission to use any existing clinical material and quote particulars of the manic-depressive patients and also for his frequent help and opinion in arriving at a correct diagnosis.

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Reports of Cases.

A SPLINT FOR THE TREATMENT OF WIDE DEPRESSED NASAL BRIDGES.

By A. B. K. WATKINS, M.S. (Lond.), F.R.C.S. (Eng.),
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THE splint described is a modification of Carter's splint which will be found described and illustrated in Phillip's "Diseases of the Ear, Nose and Throat."

The principle of the splint is shown in Figure I. Two vulcanite rods, *AA*, lie in the nasal passages along their anterior walls. The legs, *BB*, of the splint form an arch

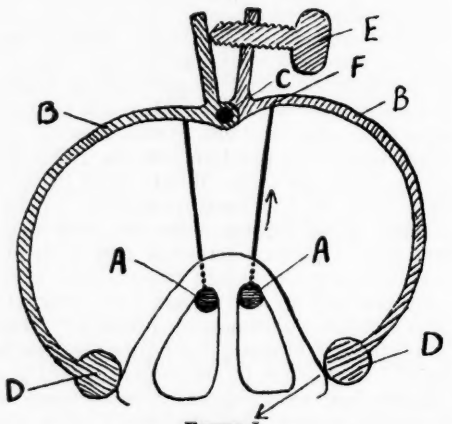

FIGURE I.
Diagram Showing Principle of Splint.

over the nose and are hinged together at *C*. The end of the legs terminate in the more or less parallel rubber covered rods, *DD*. These rods can be approximated by screwing up the thumbscrew, *E*. The middles of the rods, *AA*, are attached to silk ligatures which are passed by needle through the mucosa, cartilage and skin of the nose and are attached to the body of the splint at *F*.

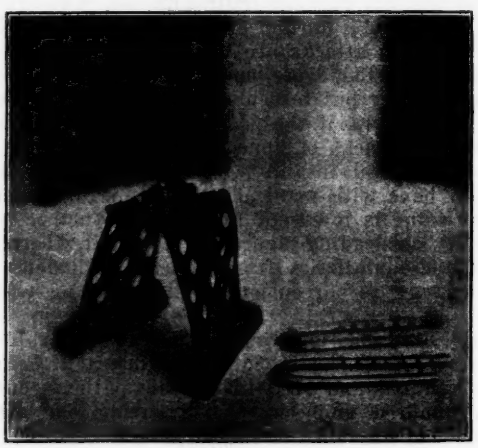

FIGURE II.
Author's Splint.

FIGURE III.
Showing Method of Exerting Traction on Splint.

In use the nasal bones and, if necessary, the nasal process of the superior maxillæ are mobilized with Asche's forceps or by osteotomy. The ligatures from the rods, *AA*, after being passed with a curved needle are attached to the splint and drawn tight. The thumbscrew is now tightened when the sides of the nose are pinched in. The triangular cross section of the nose tends to make the whole splint slip forwards. This pulls the rods, *AA*, forwards and helps to raise the nasal bridge. It must be understood that the splint is to be used for retentive purposes only after the parts have been thoroughly mobilized. If any degree of tension is used failure is certain.

The disadvantage of Carter's splint is that the nose has to be perforated by the silk threads and to overcome this I designed the splint shown in Figure II. It will be seen that the only real difference is that the intranasal splints are metal and doubled on themselves into U's and the screw is in a different site. One limb of each U is inserted into each nostril. Traction is then taken from the middle of the external limb as shown in Figure III.

FIGURE IV.
Showing Patient Before Operation.

FIGURE V.
Showing Patient at Completion of Treatment.

The following case illustrates the use of the splint.

A.A., aged twenty-three years, was admitted on March 27, 1925, to the Newcastle General Hospital. He gave a history of having done much amateur boxing; he had given this up, but not until much nasal trauma had resulted. He had a very wide, flattened nasal bridge and absolute nasal obstruction; the condition is shown in Figure IV. At the first operation a triple osteotomy of the nasal bones and of the nasal processes of his superior maxillæ was performed. The parts were then thoroughly mobilized with forceps and the splint described applied. It was only necessary to leave the splint in place until March 30, 1925. There was a mild degree of sepsis of the lateral osteotomy wounds upon which pressure had been made with the rubber ends of the splint, but this rapidly cleared up. In future such osteotomies will be done by the endonasal route. On April 4, 1925, he was discharged.

On June 25, 1925, he was readmitted very much improved as far as the flattening and width of the bony ridge were concerned. The operation scars were scarcely visible. However, there was almost as much nasal obstruction as before and the depression of the cartilaginous anterior part of the nose still existed.

On June 26, 1925, his septum was resected and adhesions between this and the lateral wall of the nose divided. Such resections in a boxer are permissible only after he has decided to fight no more, for after the operation the same amount of trauma causes greater deformities.

On July 3, 1925, and subsequently rib cartilage grafts were inserted to raise the depression below the bridge.

Figure V. shows the patient at the completion of treatment. He now has a good airway and a narrower nasal bridge than would have been the case had no splint such as I have described, been used.

AN UNUSUAL CASE OF INTESTINAL OBSTRUCTION.

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G.G., a male, aged forty-four years, was admitted to the Ballarat Hospital complaining of severe abdominal pain and vomiting.

The illness commenced at 1 a.m. on the day of admission with severe upper abdominal pain. This was attributed by the patient to eating too heartily of Irish stew for supper. The pain got progressively worse during the early hours of the morning, but eased at daybreak.

He was seen by his medical adviser at 11 a.m., when he complained of abdominal pain which was not very severe. The temperature and pulse were normal and the patient did not seem very ill. At three p.m. the pain became very severe and was referred to the umbilical region. Vomiting commenced shortly afterwards and the initial vomitus was of a dirty brown colour and offensive.

He was again seen by his medical attendant at 7.30 p.m. and in the interim his pain had become worse, the vomiting more severe and his general condition very poor. The abdomen was rigid.

The patient was removed at once to the Ballarat Hospital. On arrival his condition was very bad. The abdomen was rigid all over and was slightly distended. A mass, dull on percussion was felt to the right of the umbilicus.

Examination of the heart showed right cardiac dulness one finger's breadth to the right of the right border of the sternum. The sounds were rapid, irregular and of poor quality. There were presystolic and systolic bruits audible at the mitral area.

The vomiting was now very frequent and definitely fæculent.

The patient died before an operation could be performed.

Post mortem examination revealed an intestinal obstruction due to a large segment of orange peel becoming impacted in the small intestine. The cause of the obstruction was nearly half a small orange with the rind intact. This had become folded to form a sausage-shaped mass about six centimetres long and two and a half centimetres in diameter. One end had become impacted in a short Meckel's diverticulum (about five centimetres in length and of the same lumen as the small bowel) causing the main mass to swing round like a gate closing the lumen of the bowel.

It was afterwards ascertained that the patient had bought some oranges between eight and nine o'clock on the previous evening and had eaten one on the way home.

Reviews.

AMPUTATIONS.

DR. T. G. ORR'S little book, "Modern Methods of Amputation," is a sound, useful and satisfactory presentation of the subject.[1] He deals in turn with all the ordinary amputations, giving his reasons for adopting some methods and rejecting others. While there are some points debatable and in regard to which we are inclined to adopt another view, the debatable points are few and we are in general agreement. There is a special chapter describing the cinematiplastic procedures that have been recommended, but the author does not claim to have any personal experience in these amputations. The final chapter is an interesting one on artificial limbs. There are very numerous illustrations.

CLINICAL NEUROLOGY.

THE fact that Dr. Monrad-Krohn's book on methods for examining the nervous system has run to a third edition speaks for itself.[2] As a manual it appeals for many reasons. It is the fruit of ripe experience, handy and concise and covers such a wide field that the reader, seeking information on any particular method of examination or test, will surely not open the book without finding what he wants. And as proving the author's clinical knowledge more than a few signs are mentioned which are not to be found in ordinary textbooks, for example, in *paralysis agitans* "when the patient is asked to look to the right or left the eyes are moved *ad maximum* and the head very little or at least very slowly" (rotary neck rigidity). Again, regarding facial paresis, "it is commonly found in organic lesions such as hemiplegia, that whilst the patient's voluntary movements are decidedly paretic, yet the emotional innervation (as evidenced by the spontaneous smile) does not betray any paresis and may even be accelerated and exaggerated," a dissociation which is often bewildering to the inexperienced. And further, the account of the abdominal reflexes is based on much original observation. Although the author is a Norwegian, he was trained in British schools of neurology, also his language is clear, only here and there do we find unfamiliar expressions, as when the term "peripheral paralysis" instead of "lower neurone paralysis" is applied to certain manifestations of spinal lesions.

Features new to this edition are a chapter upon the pharmacological tests of the vegetative nervous system, accounts of the pilomotor and postural reflexes and a number of added illustrations.

DISORDERS OF DIGESTION.

PROFESSOR HUGH MACLEAN'S "Modern Views on Digestion and Gastric Disease" is the most recent addition to the series of medical monographs being issued under his editorship.[1]

Its aim, to provide a general account of the physiological processes involved in the preparation of our food for absorption and to indicate the main principles on which modern treatment of gastric ailments rests, has been most successfully achieved. Anatomy and physiology are first considered. The principles and technique of the fractional test meal and the interpretation of its results are then briefly summarized.

In a later chapter the methods of chemical examination of gastric contents and fæces are detailed. The rest of the book deals with matters of immediate clinical importance. The symptoms of the chief gastric diseases are compared and contrasted and the differential diagnostic points elucidated.

Professor Maclean puts forward convincing figures in support of his claim that early diagnosis of carcinoma of the stomach can be accomplished by means of the test meal. Radiological examination is fully discussed and some excellent illustrations of gastro-intestinal lesions given. The final chapter on treatment is concise, sound and practical.

Professor Maclean's book cannot be too highly praised. Though dogmatic in tone, it is so sound that little criticism of the views he puts forward, can be offered. The account of the organic diseases of the stomach is masterly.

The functional disorders of digestion, the most difficult aspect of the subject, have not been handled quite so successfully and we feel that they might have been discussed a little more fully. But this is a minor criticism.

The general practitioner to whom the book is specially addressed, and the senior student will look to it gratefully for definite guidance in a difficult field of medicine and even the specialist in gastro-intestinal diseases, though he may occasionally rebel at its dogmatism, will find in it much food for thought.

[1] "Modern Methods of Amputation," by Thomas G. Orr, A.B., M.D., F.A.C.S.; 1926. St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 117, with illustrations. Price: $3.50 net.

[2] "The Clinical Examination of the Nervous System," by G. H. Monrad-Krohn, M.D. (Oslo), M.R.C.P. (London), M.R.C.S. (England), with a Foreword by T. Grainger Stewart, M.D., F.R.C.P.; Third Edition; 1926. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 217, with illustrations. Price: 7s. 6d. net.

[1] "Modern Views on Digestion and Gastric Disease," by Hugh Maclean, M.D., D.Sc., M.R.C.P., 1925. London: Constable and Company, Limited. Sydney: Angus and Robertson, Limited. Demy 8vo., pp. 170, with illustrations. Price: 14s. 6d. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 23, 1926.

The Prevention of Disease.

In the summary of the proceedings of the Federal Committee of the British Medical Association in Australia published in our issue of September 18, 1926, reference was made to a conference of the Ministers of Health of the Commonwealth and of the several States held for the purpose of considering the recommendations included in the report of the Royal Commission on Health. This conference took place on July 21 and 22, 1926, but as it was an interdepartmental conference, the discussions were regarded as confidential. The resolutions adopted have now been made public by their publication in the September issue of *Health*. In view of the importance of their findings, we reproduce the resolutions on another page of this issue. It will be noted that in addition to the Ministers and their representatives the responsible medical officers of the health departments of the Commonwealth and of four States were present. The Commissioner of Health of Queensland was not able to attend; his place was taken by the Assistant Under Secretary. The position of chief medical officer of Tasmania is at present unfilled and consequently no representative could be sent. Notwithstanding the fact that conferences of the representatives of the health authorities of the several States with the Director-General of Health of the Commonwealth in the past have at times proved futile on account of the failure of one or other State to adopt the measures accepted at the meetings, it may be assumed that progress has been achieved on this occasion. In the first place the resolutions have received endorsement of six Ministers of Health and one Secretary of the Department of Public Health representing the Minister. In the second place the resolutions may be regarded as an acceptance of the proposal of the Royal Commissioners that a body should be established to be known as the Federal Health Council and that this body should consist of the Director-General of Health, two other representatives of the Commonwealth and a financial expert of the Commonwealth Government and the chief medical officer of each State. The functions of the Federal Health Council are defined in the report as follows: To devise measures for cooperation of Commonwealth and States and of States with States and to promote uniformity in legislation and administration where advisable. The conference, after having accepted this proposal in its entirety, determined to refer to the Federal Health Council the model scheme outlined in the report for the prevention of disease, the question of the adoption of uniform measures of control of food and drugs, the recommendations concerning maternal hygiene, the provisions for the promotion of child welfare, the general principles on which industrial hygiene should be based, and the plan for the cooperation of the practising portion of the medical profession with the public health authorities. The first resolution deals with the collection and tabulation of information and does not call for comment in this place at present. The resolution referring to the control of venereal diseases takes the form of a general approval of the recommendations of the Royal Commissioners with the possible exception of the suggestion that marriages should be made contingent on the production of sworn statements to the effect that neither of the contracting parties is suffering from a "contagious or infectious disease." The conference endorsed the proposals of the Royal Commissioners in regard to research. All the other resolutions come under the category of reference to the Federal Health Council.

It is evident that the next step must be taken by the Commonwealth Department of Health. The institution of the Federal Health Council is necessarily a task for the Commonwealth Government. It is reasonable to hope that the effect of this new body will be to remove the last traces of interstate differences, to substitute for these differences a spirit of honest collaboration for the improvement of the health of the people of Australia. Uniformity of action should be the outcome, since it is self-evident that only slight variations in the application of prophylactic measures are needed to meet the requirements of various localities. The Com-

monwealth Department of Health will be called upon to undertake a leading part in the endeavour to control preventible disease, for it cannot be held that the combating of infections can be attempted with good prospects of success by one State without the adoption of similar measures in other States. No State is asked to relinquish its rights, but all are expected to introduce legislation based on common principles and all are expected to administer the health laws on a uniform plan of action. If the Federal Health Council justifies its existence, Australia will derive the benefit of a sound scheme for the prevention of disease and for the maintenance of a high standard of health. It will mean a reform in public health administration, perhaps a revolution. It is true that the Federal Health Council is to be an advisory body, but advisory bodies can assume very important functions in the moulding of policies.

The practising part of the medical profession will be required to take an active part in the campaign. While the Federal Health Council will coordinate the efforts of the health authorities, the primary work of attacking infection at its source will be in the hands of the general practitioner. If the profession is wise, the general practitioner will mould the policy of the health authority by revealing his initiative and by devising with his colleagues a sound method of procedure in the direct attack on disease.

Current Comment.

SYMBIOSIS OF MICROORGANISMS.

It has been known for some years that two organisms, living "in symbiosis or artificially mixed together" may produce gas fermentation in certain carbohydrates, though neither of them is capable of doing so alone. Dr. Aldo Castellani found that baker's yeast is not a pure culture of yeast. It is generally composed of one or more species of yeast with one or two varieties of bacteria. He found that whole yeast ferments a wider range of carbohydrates than is fermented by any one of the saccharomyces and bacteria composing the sample. Moreover, he obtained fermentation of maltose with production of gas by mixing artificially non-maltose fermenting saccharomyces, isolated from a sample of baker's yeast, with a bacillus isolated from the same sample, the bacillus being one which produced acidity in maltose and several other sugars, but did not produce gas in any other substance. In 1925 Dr. Castellani published the results of a preliminary investigation into the biochemical characters of bacteria and indicated that the account of a more complete investigation would appear at a later date.[1] He mixed *Bacillus typhosus*, an organism which produces acidity but not gas in maltose, mannitol and sorbite, with *Bacillus morgani*, an organism which produces neither acidity nor gas in these substances. He pointed out that it would be expected that such a mixture would merely produce simple acidity. The result, however, was the production of both acidity and gas. He expressed the opinion that such a phenomenon seemed to depend to some extent on the fermentative powers of the second germ on certain other carbohydrates. For instance not all bacilli, inert on mannitol, will cause production of gas in that substance when added to *Bacillus typhosus*. He thought that a condition for the occurrence of the phenomenon was the capability of the added germ of producing acidity and gas in glucose. At the same time not every organism, producing gas in glucose and neither acidity nor gas in mannitol, will cause the production of acid and gas in mannitol when added to a producer of simple acidity such as *Bacillus typhosus*. Dr. Castellani concluded that the subject was very obscure and required much further investigation.

He has recently published a further communication on the same subject.[2] After going over some of the ground in his previous article, he describes many instances in which organisms produced gas when grown on either mannitol, maltose or adonite, although when grown alone no gas was formed. In some instances neither gas nor acidity was produced when the organisms were grown alone. The groups of organisms are as follows: *Bacillus dysenteriæ* (Flexner) and *Bacillus morgani; Bacillus dysenteriæ* (Flexner) and *Bacillus proteus; Bacillus typhosus, Bacillus morgani* and *Bacillus proteus; Bacillus coli communis* and *Bacillus kandiensis; Staphylococcus aureus* and *Bacillus morgani; Staphylococcus aureus* and *Bacillus proteus; Streptococcus pyogenes* and *Bacillus morgani; Streptococcus pyogenes* and *Bacillus proteus.* In explanation he refers to the view of Harden and Schütze that the phenomenon is dependent on the fact that one of the organisms produces certain substances which are fermentable by the other organism.

Dr. W. L. Holman and Dr. D. M. Meekison have also discussed this question.[3] They begin by attacking the use of the word symbiosis in this connexion. They hold that symbiosis suggests mutual benefit and they are not convinced that this plays any part in the phenomenon. Dr. Castellani on the other hand divides symbiosis into two grades, symbiosis *sensu lato* and symbiosis *sensu stricto*. The former of these terms is used by him to indicate the living together of two organisms in close association, the association not being detrimental to either of them. The latter indicates that the association is not only

[1] *The British Medical Journal*, October 24, 1925.
[2] *The Journal of Tropical Medicine and Hygiene*, August 2, 1926.
[3] *The Journal of Infectious Diseases*, August, 1926.

not detrimental, but mutually beneficial. Dr. Holman and Dr. Meekison prefer the term synergism. They hold that it defines merely what occurs.

In connexion with this subject it is necessary to refer to what has been termed the inhibitory phenomenon. This is the reverse of the phenomenon under discussion. Theobald Smith and D. E. Smith found that paratyphoid bacilli inhibited the fermentation of lactose by *Bacillus coli communis* added later on. They found that the grade of inhibition depended on the age of the cultures of the paratyphoid bacilli and they also found that living, active bacilli were necessary. It has been suggested that the inhibitory substance is some metabolic product of the paratyphoid bacilli, possibly an enzyme. Dr. Castellani merely mentions the inhibition phenomenon in passing, but Dr. Holman and Dr. Meekison regard it as having a direct bearing on the gas production phenomenon. They hold that an intermediate substance is formed by the breaking down of the carbohydrate and that in the gas forming phenomenon the two bacteria absorb the intermediate substance in proportion to their activity and, acting on it, produce acid or acid and gas; if the environment is unfavourable, the gas producing function may be in abeyance. In the inhibition phenomenon, however, the interference in gas production may in their opinion have depended on the absorbing activity by the paratyphoid bacilli of the intermediate product set free from the medium by *Bacillus coli* and its metabolism by the paratyphoid bacilli.

Dr. Holman and Dr. Meekison conclude that for the phenomenon to be manifest the bacteria must be living in close association, one of the pair must be capable of splitting the test substance and forming acid and the other must be capable of forming gas from monosaccharides. The activity of the bacteria and the stage of growth are important considerations. The two bacteria may be present in the mixture and yet no gas may be formed. If the acid-producing organism finds the medium so favourable that it can multiply up to a certain stage, the gas-producing organism will fail to produce gas. In other words the inhibition phenomenon takes place instead. It is thus obvious that various grades of the phenomenon of gas production can be obtained. Dr. Holman and Dr. Meekison did not test the effect of heat, but refer to the findings of Theobald Smith and D. E. Smith in this connexion. These authors found that the inhibition phenomenon was not entirely destroyed when the bacteria were killed at the minimum temperature, but that it gradually disappeared if the recently killed bacteria were incubated for a day or two or if the temperature used for killing was raised to 100° or over. It is suggested that the bacteria recently killed by heat are still sufficiently absorbent to take up enough of the intermediate substance to lessen the inhibition and that this absorbing power is lost as the cell wall is more and more altered by heat and age. Dr. Holman and Dr. Meekison point out that it is difficult to prove or to disprove this view, but that the same principle of physiological activity has been found of importance in such studies as those of the bacteriophage. Another factor of great importance in the gas production phenomenon is the hydrogen ion concentration. A chapter could be written on this part of the work alone. One example of the variation in acidity alone will be given. Gas production by *Bacillus coli communis* was found to be checked by the increased acidity from the streptococci and the two bacteria reached their maximum acidity at different periods in their growth curves. It was found that the process could be restarted by the addition of alkali when it was held in check by the acid present.

There seems to be some confusion between the behaviour of bacteria grown outside the body in the presence of a chemical substance that never occurs within an animal organism and that may be regarded as an artificial environment, and the behaviour of pathogenic organisms growing within the body. The sugar reactions are convenient methods of applying rough classifications of certain classes of bacteria. Symbiosis is necessarily a biological phenomenon, the influence of the growth of a second organism on the effect of the primary infecting agent. Mere association of two or more kinds of bacteria in one living host does not constitute what is usually regarded as symbiosis. Symbiosis probably depends on the physiological effect of a combination of bacterial products. This may be of the nature of a true chemical reaction or it may be a physical property. In either case the physiological effects of the products of the primary infecting organism will be modified by the change in those products by the antagonism or anchoring of the products of the second bacterium. There is no evidence at all of a process analogous to that which occurs when a second bacterium is superimposed on an artificial culture of a primary organism in one of the carbohydrate media. The workers named above postulate the formation of a new substance of an enzymic nature out of the dissociation of the carbohydrate and suggest that this new substance absorbs acid and gas in the inhibition process. The dissociation products of carbohydrates are simple substances. On the other hand it is known that enzymes are elaborated in the process of dissociation of bacterial protein or as we should prefer to have it, that the atomic arrangement of the complex molecule of protein may become modified in such a manner that enzymic action is manifested. It appears to be more probable that the non-appearance of acid and gas or of either acid or gas is caused by the adsorption (not absorption) of these products on to the surface of split proteins. The changes noted in the behaviour of the associated bacteria toward the sugars *in vitro* bear a striking resemblance to the mutations which have been effected in pure cultures of bacteria when grown repeatedly on unusual artificial media. Unless some direct evidence is produced, it would be unsafe to assume that these changes take place within the body when a second organism is added to an existing infection and modifies the manifestations of the primary infection. The subject is one of real importance, since its application to disease problems must be an extensive one.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Rheumatic Heart Block.

G. A. Sutherland (*Archives of Disease in Childhood*, February, 1926) reports the history of a patient with severe rheumatic heart block who was treated by adrenalin. A schoolboy, seventeen years old, was noticed by a brother at 3 a.m. to have a fit. The patient had just returned to bed after having risen to take a drink of water. From 3 a.m. to 5 a.m. he had a series of fainting attacks and at 5 a.m. another fit. A doctor was summoned and found that numerous fainting attacks occurred in which the patient turned pale, but did not lose consciousness. He complained of feeling faint. In the more severe attacks he became pale, the legs and arms were fixed and he lost consciousness; some twitching of the limbs and face occurred, he breathed deeply, the face became flushed and he recovered consciousness. The severe attacks became more frequent at midday. The boy was seen by the author at two o'clock of the same day. He was feeling tired and exhausted. The pulse was at times steady and regular at eighty per minute and at other times irregular with pauses of varying length. During twenty minutes the pulse stopped twice for long intervals and each time a "fit" occurred, clearly an example of the Stokes-Adams syndrome. In regard to the cause of this attack there was not much evidence to be obtained. Two days previously the boy had returned home feeling tired with a temperature of 37·5° C. (99·5° F.). Next day he felt better. Apart from this he complained of no symptoms. The diagnosis of severe heart block was made and 0·24 mil (four minims) of adrenalin solution (1 in 1,000) was given hypodermically. The injection was repeated the same evening; two similar injections were given on the two following days and one injection daily for the next two days. From the time of the first injection there occurred no more attacks of the Stokes-Adams syndrome and very few fainting attacks. The progress of the condition threw a flood of light on the nature of the sudden onset. His temperature rose to 38·3° C. (101° F.). He sweated profusely during the night and complained of pain in the left ankle which was found to be swollen and tender. He passed through an ordinary attack of rheumatic fever and in a week was convalescent. Six months later he was taking his full share in all the school sports. In this case the conducting tissues of the heart were involved at an early stage of the rheumatic infection possibly with other myocardial involvement, but in areas which were silent as regards symptoms. The author suggests that there was some toxic disturbance in the conducting tissues, possibly some œdema or vascular disorder and that the local disturbance was temporary. When auriculo-ventricular dissociation with a very slow ventricular rate is present, anything which increases the rate of the ventricles, would be helpful. This is what adrenalin seems to do either by stimulation of the sympathetic or by direct stimulation of the ventricles. The result is that a tendency to prolonged standstill of the ventricles from which come the dangerous symptoms, is checked as long as the effect of the adrenalin lasts.

Rickets.

Agnes H. Grant (*American Journal of Hygiene*, March, 1926) describes experiments showing the effect of lowering both the antirhachitic vitamin and the calcium in the diet of the mother upon the development of rickets in the young. Although rickets is primarily due to a lack of the antirhachitic vitamin and will develope when this is the only deficiency in the diet, the presence of a second deficiency hastens the onset of the disease and makes its progress more rapid. Lowering the calcium content of the diet without at the same time lowering the phosphorus disturbs the normal balance between these salts and seriously affects the amount of vitamin necessary to insure normal bone growth. When the mother's diet is deficient in calcium, large amounts of the vitamin are necessary to maintain the calcium-phosphorus ratio in her milk and when the antirhachitic vitamin is also deficient, the effects are very serious. The young reach weaning age with very small vitamin reserves and with almost no resistance to the effects of deficient diets. The double deficiency of vitamin and calcium are even more serious when the deficiency of vitamin has been prolonged. In this case the young sometimes develope signs of rickets before they reach the weaning age, before they have had any food other than their mother's milk.

The Nutritional Requirements of Nursing Mothers.

Agnes H. Grant and Marianne Goettsch (*American Journal of Hygiene*, March, 1926) discuss the nutritional requirements of nursing mothers. Using rats in their experiments, they investigated the effects of a deficiency of the antirhachitic vitamin only in the diet of the mothers upon the development of rickets in the young. Studies of normal young have shown that the rats from mothers that are kept on a carefully planned laboratory diet, grow to maturity before their inherited reserves of vitamin are depleted, even though they are given a diet which is wholly deficient in vitamin from the time of weaning and are kept in darkened cages. Partial deficiencies which are extended over a long period of time, bring to light defects that total deficiences cannot show. The authors found that partially depriving the mother rats of the antirhachitic vitamin and forcing them to raise several litters in darkness, lowers their reserve of this factor before it interferes with their ability to raise their young. In the absence of light the diet furnishes the only source of the antirhachitic factor and when the vitamin content of the diet is lowered, the mother rats cannot recuperate fully after raising their first litters. Subsequent litters deplete their reserves so far that fourth and fifth litters have almost no inherited reserves of the antirhachitic vitamin, although they have sufficient vitality to grow to maturity. When all the litters are kept under the same conditions and on the same deficient diet, the gradual lowering of the vitamin reserves of the mothers can be accurately traced by the steadily decreasing resistance of the young to rickets. The authors conclude that rickets is primarily due to a lack of the antirhachitic vitamin and will develope when a lack of this vitamin is the only deficiency in an otherwise adequate diet. The diet of the mother is an important factor in increasing or decreasing the resistance of the young to the effects of diets which are deficient in the antirhachitic vitamin.

Streptococcus Pneumonia in a New-Born Infant.

Charles M. O'Connor (*Buffalo General Hospital Bulletin*, July, 1926) reports a case of streptococcus pneumonia in a new-born infant, probably the result of antenatal infection. The infant, a female, was born by normal delivery. She cried immediately after birth and for the first twenty-two hours appeared perfectly healthy. Her temperature then rose to 38·9° C. (102° F.), cyanosis appeared together with shallow and rapid respirations with very feeble cry. Examination of the lungs and heart revealed no definite abnormality. On the assumption that the child was suffering from intracranial hæmorrhage, it was given ten cubic centimetres of blood subcutaneously. The condition became rapidly worse and the infant died seven hours later. The only relevant point in the mother's history was that towards the end of her pregnancy she was found to be suffering from inflammation of the nasal sinuses. She was treated for this and it was stated that all the sinuses including the ethmoidal were severely involved. On the afternoon of her confinement her temperature rose to 38·30° C. (101° F.). On the third day it rose to 39·5° C. (103·2° F.), but gradually subsided. The rise in temperature was regarded as the result of the sinus infection. On *post mortem* examination of the baby the cranial cavity was found to be free from blood and the tentorium intact. In the abdominal cavity about fifty cubic centimetres of clear fluid was found. The serosa especially in the region of the mesentery was diffusely thickened and around the ascending colon a few small fibrous bands could be traced running to the root of the mesentery. In the chest cavity on both sides there was a considerable excess of fluid which contained numerous fibrin flocculi. The lungs appeared

swollen, the pleura in most places had lost its lustre and in some places, especially on the right side, it was covered with a fine fibrinous exudate. On section both lungs manifested numerous coalescent areas of consolidation of greyish-red colour. The lower lobe of the right lung appeared almost completely hepatized. Cultures from the lungs, spleen and liver yielded among other bacteria, a large number of colonies of hæmolytic streptococcus. The question of the origin of the pneumonia is discussed. Careful examination revealed no evidence of infection of the umbilical cord. The pneumonia seemed to be interstitial in character, involving probably the pleura in the first instance and propressing along the interlobular septa to the interior of the lung. The larger bronchi were not involved in the process. In the presence of the findings, pointing to an infection starting from the pleural cavity, an infection which cannot be regarded otherwise than as a blood infection, the author concludes that its origin was intrauterine, probably through the placenta.

ORTHOPÆDIC SURGERY.

Congenital Dislocation of the Hip.

BENJAMIN P. FARRELL, HERMAN L. VON LACKUM AND ALAN DE F. SMITH (*The Journal of Bone and Joint Surgery*, July, 1926) make a final report of the condition of three hundred and ten patients suffering from congenital dislocation of the hip treated at the New York Orthopædic Dispensary and Hospital from October, 1900, to October, 1920. The most recent patient in the series was treated more than five years ago. In eighty-five patients the condition was bilateral, making a total of three hundred and ninety-five hips. Of these two hundred and sixty-six were followed over a period long enough to allow determination of the end result. The Hibbs table was used in the majority and four dislocations were reduced by open operation. It is now the custom in this hospital to resort to the latter method of treatment in a much larger proportion of cases. In the two hundred and sixty-six cases that were traced, one or both hips remained in the acetabulum in only one hundred and three. Among the 61% of femoral heads that did not remain in place, were forty cases of anterior transposition. These gave fairly good functional results and raised the proportion of satisfactory results to 54%. Of the patients 85·2% were females and in 65% the left hip was dislocated. The authors lay stress on the importance of the age of the patients at the time of reduction and emphasize this in an analysis according to the age group. In patients under five years of age, 53% of dislocations were reduced with a good result. In the second group of those at five and six years 43% were reduced with a final good result. Only 23% of cures was obtained when the patients were in the seventh and eighth year period. Torsion of the shaft of the femur caused anteversion of the neck of more that 20° in one hundred and eighty-eight of the hips or 47% of the series. In the opinion of the authors anteversion is most important and is one of the prime factors producing dislocation as well as causing redislocation to occur. An attempt was made to correct anteversion in thirty-five instances. Thirteen of the femoral heads subsequently remained in place and twenty-two of them did not. Four patients were subjected to open operation. All were definitely improved, but limitation of movement remained in three. A perfect anatomical result judged by X ray examination was found only in three instances. In others there was more or less irregularity of the acetabulum. The functional result is usually better than can be expected from the X ray appearances. After reduction the hips were immobilized in plaster of Paris in the position in which they seemed most suitable. This was from 60° to 90° abduction. The period in plaster casts varied from one and a half to fifteen months. The authors believe that prolonged immobilization is unnecessary in suitable cases and in others it accomplishes very little. The most important reasons for failure appear to be age, anteversion of the neck, bony abnormality of the acetabulum and of the head and neck of the femur and contracture of the cotyloid ligaments.

Tendon Transplantations.

L. MEYER (*Journal of Bone and Joint Surgery*, April, 1926) deals with tendon transplantations for division of the extensor tendon of the fingers. He points out that lesions in the finger do not give good results, but when the lesion is in the hand, a higher percentage of cures can be expected. The *extensor communis digitorum* is used commonly to replace the injured extensor tendon. In performing the operation local anæsthesia is preferable. Care should be taken in operating not to place the incision over the tendon that is to be spliced. The author uses braided silk in a fine straight cambric needle with a calyx eye. Fingers should be kept immobile in extended position for eight days, when active movements should be begun. At the end of four weeks the range of movement is about 75% of the normal.

Disinfection of Septic Joints.

F. J. COTTON (*Journal of Bone and Joint Surgery*, April, 1926) holds that simple drainage of septic joints is not sufficient in most cases, but that proper disinfection should also be part of the treatment. He describes his technique as simple. In the knee the pouch of the quadriceps is entered through a small incision on the outer side. Through this opening the knee is washed out for fifteen minutes with a normal salt solution to which has been added one in fifteen thousand corrosive sublimate. When the washing is finished, the capsule is sewn up again. The outer wound is left open and an alcohol dressing applied. The patient may have a rise in temperature for two or three days after which the joint returns to normal. Movement is allowed on the tenth day. Case histories are given.

An Unusual Type of Paralytic Abduction Deformity of the Hip.

L. MEYER (*Surgery, Gynecology and Obstetrics*, March, 1925) draws attention to an unusual type of paralytic abduction deformity of the hip and suggests an operation for its cure. The deformity was due to the unopposed action of healthy abductors. The pelvis was tilted down on the same side and there was a consequent right lumbar scoliosis. The operative treatment consisted in dividing the *fascia lata*, chiselling the tip off the trochanter and incising the capsule near its attachment to the neck of the femur. Prevention of the recurrence of the deformity was effected by making use of a strip of *fascia lata*. This was carried across the front of the thigh and stitched to the inguinal ligament and the spine of the pubis. There was no attempt to replace the tip of the trochanter and the clinical result was good.

The Significance of the Accessory Tarsal Scaphoid.

I. ZADEK (*Journal of Bone and Joint Surgery*, July, 1926) draws attention to the importance of the accessory scaphoid. He gives details of an operation upon a girl, eleven years old. At operation a most unusual condition was found. The tendon of the *tibialis posterior* was found inserted entirely into the navicular and did not extend beyond this bone. The accessory scaphoid was completely enclosed in a conical expansion of the *tibialis posterior* tendon. The author says that it seems likely that the presence of an accessory scaphoid might be suspected, if it were found that the tendon in the *tibialis posterior* did not pass beyond the scaphoid in association with the bony prominence in the region of the tubercle of the scaphoid. The author has operated upon two other patients and found identical conditions. All these patients presented symptoms of an ordinary weak foot. The author contends that the importance of the accessory scaphoid lies not in its presence or absence *per se*, but in the fact that it indicates an abnormal condition of the *tibialis posterior* muscle.

A Traction Loop for the Foot.

JOHN N. PORTER (*Journal of Bone and Joint Surgery*, April, 1926) describes a method for applying traction to the foot and leg. The material used is a filet about one and a half metres (five feet) long and 3·75 centimetres (one and a half inches) wide made of two layers of canvas. There are figures illustrating the application of this filet and the author holds that considerable traction can be exercised without constriction or discomfort.

British Medical Association News.

SCIENTIFIC.

A meeting of the South Australian Branch of the British Medical Association was held at the Lister Hall, Hindmarsh Square, Adelaide, on July 29, 1926, Dr. H. H. E. Russell, the President, in the chair.

Tuberculosis.

Dr. F. S. Hone read a paper entitled: "The Prevention of Tuberculosis" (see page 539).

Dr. H. W. Wunderly read a paper entitled: "Some Notes on the Scientific Treatment of Pulmonary Tuberculosis with Reference to the Pathological Types and Their Diagnosis" (see page 543).

Dr. J. W. Browne agreed with Dr. Hone that much could be done in the way of prevention and much more than was done at present. The methods were the same as in other infective diseases, namely isolation, as far as possible, of patients with active tuberculous disease and supervision, as far as possible, of contacts. With adequate isolation there would be less possibility of massive doses of infection, which were in most cases necessary for the lighting up of active disease owing to the previous fortuitous inoculations of the majority of adults in civilized communities. With regard to contacts the fact that nearly 50% of tuberculous patients had a family history of tuberculosis, should be considered as a severe comment on the methods of prevention employed in such families.

With regard to prophylactic vaccination, Dr. Browne recalled the fact that in a paper read before the Branch in October, 1921, he had stated his belief in the possibility of such prophylaxis being discovered and had detailed some of the experiments of Dr. Nathan Raw in this regard. He had pointed out that Calmette's vaccination must be performed before the infant had been sensitized or infected by a fortuitous dose of tubercle bacilli. With regard to treatment he stressed the absolute necessity of prompt recognition of early cases, if results of any value were to be obtained. To illustrate this point he gave the statistical results of an investigation made in the year 1919 and detailed in the annual report of Kalyra Sanatorium for that year. From that report it appeared that out of the seven hundred patients who had passed through the sanatorium between the years 1906 to 1911, some 50% were dead within two years of the onset of their illness, 65% died within five years and 70% within ten years. These seven hundred had been in various stages of disease on admission, some in early and some in advanced stages. When patients in the early stages only were considered, the results of treatment were much better. About 70% of such patients had their disease arrested at the sanatorium and of these probably more than half remained permanently well.

Dr. Browne had not been impressed with the value of tuberculin as a method of treatment. He denied its right to be considered as in any sense a specific. The characteristic of a specific was its universality of application, in illustration of which he quoted as examples of three specifics, quinine in malaria, mercury in syphilis, "Insulin" in diabetes. According even to its advocates tuberculin could be used in quantities about which no two of them quite agreed and only in a severely limited class of case, very hesitatingly differentiated even by experts with many provisos and numerous exceptions. He gave his experience at Kalyra of tuberculin in treatment. The number of cases of arrest, during a period of five years in which tuberculin had been consistently employed, were compared with the number in the subsequent five years during which it was not employed. The use of tuberculin was found to have made practically no difference. In the King Edward VII. Sanatorium in England, Dr. Noel Bardsley some years later had made the same comparisons and had come to precisely the same concluions.

He referred shortly to the methods of chemotherapy such as the administration of "Sanocrysin" and stated that, while interesting and even promising, these methods were still in the experimental stage.

Dr. D'Arcy Cowan said that tuberculosis, especially pulmonary, once firmly established, was such a serious condition and the results of treatment were so poor, that prevention must occupy an important position in their calculations. There was no one better qualified than Dr. Hone to deal with this subject and there was nothing to add to what he had said. If it were possible to destroy all the tubercle bacilli as they had destroyed their hosts and in this respect the bovine and the human were the only sources that need attract attention, the disease would soon be controlled. This was an ideal that should always be kept in mind, but it was an ideal very difficult of attainment.

In the case of bovine tuberculosis the only real source of danger was raw milk and this could be made safe by the simple expedient of boiling before use. Boiling did not destroy its food value or vitamin content in any way. The next best thing was to diagnose infections early and by efficient treatment prevent them from becoming advanced and open forms of the disease. Tuberculosis in its early stages, when still limited by the lymphatic system, was an eminently curable disease. The natural tendency in most cases was toward cure and with early recognition of the disease and appropriate treatment most, if not all, patients would recover. In a few patients with poor resisting power who acquired a virulent infection, the course of the disease was rapid and there was no treatment of any avail. Happily these patients were not common. Apart from these most patients passed through a stage at which the lymphatic system made a more or less successful resistance against the tuberculous invader. In this stage the symptoms were indefinite and comprised chiefly tiredness, loss of weight, functional digestive disturbance, pallor and low blood pressure. These were the symptoms of a chronic toxæmia and were not confined to tuberculosis, but nevertheless, tuberculosis was probably the commonest cause of chronic toxæmia and should always be thought of if these symptoms persisted.

In this stage there were no characteristic physical signs.

The stethoscope was a perfectly good instrument if its limitations were recognized, but in these conditions it might as well be applied to the back fence for all the information that would be gained. It was in the early diagnoses that the profession failed so hopelessly. Medical practitioners appeared to think that because they found no abnormal physical signs, therefore there was nothing wrong, anæmia, neurosis, neurasthenia and "run down" were the common diagnoses. They did not recognize the pitfall of deep seated disease. And the same remarks applied almost exactly to early surgical tuberculosis. There were no characteristic signs and symptoms except perhaps an enlargement of superficial glands and even in these circumstances it was impossible to say if the adenitis was tuberculous or merely simple chronic inflammation.

As soon as it was possible to recognize definite physical signs in an organ or find tubercle bacilli in the discharges, the tuberculosis was no longer in the early stages and the results of treatment were doubtful. The patient's best chances of recovery had gone. There was only one way to diagnose these conditions and that was the tuberculin test. He did not propose to enter into details of that test. With proper care and due recognition of the indications and contraindications the test was safe and reliable. It was said that there was danger of lighting up a latent focus, of causing dissemination of the disease and of other dreadful things. In an experience of fifteen years not one untoward effect had occurred to cause more than temporary uneasiness. On one occasion slight hæmoptysis and on another slight hæmaturia had occurred following a test dose and these symptoms had very quickly subsided. Tuberculin, properly used, did not cause dissemination of the disease, but tended to prevent it. Dr. Cowan had seen at least a dozen deaths from tuberculous meningitis in which no tuberculin whatever had been given. On the other hand in all his patients treated by tuberculin tuberculous meningitis had not once occurred. Three things might happen after a subcutaneous injection. In the first place there might be no reaction whatever; in the absence of cachexia or advanced disease this meant absence of active tuberculosis. This had occurred in approximately 50% of cases in which tests were made. In the second place there

might be a febrile reaction with focal signs and symptoms due to active hyperæmia at the site of disease. This meant active tuberculosis. This had occurred in approximately 25% to 30% of his patients. In the third place there might be a febrile reaction with no recognizable focal symptoms or signs. This meant tuberculous infection, but did not indicate the site or condition of the lesion whether active or not. In these cases the final conclusion must be arrived at on other grounds. This had occurred in 20% to 25% of his cases and was the only weak point in the test.

This test was destined to play a great part in the prophylaxis of tuberculosis. The wider use of tuberculin in diagnosis was essential. It had never let him down badly; without it the early diagnosis of tuberculosis was pure guesswork and the members of the medical profession were notoriously bad guessers. Missed early diagnoses were so common as to be almost a rule. One patient whom he showed that night was typical. For three years the lymphatic system made a valiant struggle against the tuberculous invader. In that stage the disease was easily curable, but it was not recognized until hæmoptysis occurred and the damage was done. From then on this patient's troubles started and he had been driven to drastic measures like artificial pneumothorax and other collapsing operations. The fact that these had to be done was a confession of failure. There was nothing more depressing than the treatment of a patient in the advanced stages of tuberculosis.

The history of a doctor might be mentioned. He was one of the finest men whom one could wish to meet and his story was pathetic. In his fifth year of medicine he had become off colour, he was tired, had lost weight and suffered from functional digestive disturbances. These were the common early symptoms. He had been examined time after time by the best physicians of his city and the cause of his ill-health had not been diagnosed; it was even suggested that he was neurotic and he was urged to buck into his games and not think so much about himself. This state of affairs had continued for six years and then the patient had made his own diagnosis when he found tubercle bacilli in his sputum. His whole life had been ruined by failure to make a diagnosis and institute effective treatment at the right time. Tuberculin would have saved all this. In the early stages specific treatment with tuberculin was just as valuable as the use of tuberculin in diagnosis. In later stages such good results could not occur, but even there in certain cases very gratifying success was attained. Dr. Wunderly had dealt very ably with this subject and Dr. Cowan said that he had nothing further to add. With regard to surgical tuberculosis he would urge the surgeon to make greater use of tuberculin in diagnosis and treatment. He had recently had the pleasure of attending Robert Phillip's tuberculosis clinic in Edinburgh. The results attained there by the use of tuberculin by inunction were astonishing. The person who could go and see the results obtained in that clinic and come away unconvinced that the patient had received something from the inunction, must be very sadly lacking in his own powers of observation. The great principles in the treatment of surgical tuberculosis were rest and avoidance of mixed infection. These were just the two factors that made the treatment of lung tuberculosis so difficult. But the constitutional treatment was absolutely essential. Almost all conditions of surgical tuberculosis were secondary manifestations and eradication of these did not remove the primary focus.

Dr. Cowan had recently assisted a surgeon in the removal of some tuberculous glands from the neck. His quiet remark while searching for further glands would not soon be forgotten: "There is always one more." That put the case in a nutshell. A tiny gland removed, only the size of a sago grain, on section manifested tuberculous deposits. They had listened in that room to a long harangue by one of their members decrying tuberculin in all its forms and summing up by saying that tuberculin was a commercial vaccine and a gigantic hoax. Teaching of this sort was pernicious and the value of that opinion could be estimated when they read in the reports of a certain institution for 1921:

> No form of tuberculin has been administered in this institution since 1914. No evidence in its favour as a curative agent for pulmonary tuberculosis sufficiently satisfying to induce us to alter the opinion on its efficiency expressed in the report for 1914 has been brought forward since that time.

Again in 1925 they read:

> The report regarding them (tuberculin *et cetera*) are conflicting, and until the value of some of them has been demonstrated beyond all doubt we shall continue to use the remedy that has stood the test of time.

Opinions of this sort from persons who had not used these remedies, were useless and harmful.

In conclusion Dr. Cowan asked permission to read a letter recently received from a patient which demonstrated very well the attitude of the lay public towards this question, and unfortunately to a large extent also the medical profession. The patient stated:

> If you remember you told me I had tuberculosis and gave me a good fright. You had no right to say such a thing, as my lungs were as good as yours. After seeing you I went to my lodge doctor who rung you up and he told me not to take any notice of your finding. My trouble as I told you was my stomach, and I am suffering from severe dyspepsia. How you got your conclusions I don't know and neither does anyone else. Not having been able to work for some months *et cetera*.

This man had been seen on one occasion only; he had not allowed Dr. Cowan to complete his investigations of his condition. It was interesting to note that his dyspepsia had continued and he was unable to work (and this was twelve months later). Owing to his stupidity the cause of his ill health had not been discovered, but the odds were ten to one that he had a tuberculous infection.

The doctor who diagnosed tuberculous disease in the absence of hæmoptysis or tubercle bacilli in the sputum or other obvious signs, was looked on with suspicion both by the public and the profession and the tragic part of the business was that it was only in this stage that they had a reasonably good chance of curing the patient.

Dr. Cowan then showed several patients.

The first patient was a man, aged thirty-six years, who had been referred by Dr. Edgar Browne in March, 1924. In 1904 the patient had suffered from a nervous breakdown and albuminuria. He had seen an eye specialist who detected no retinal changes. He had been away from school for nine months and the albuminuria disappeared. In 1914 he had suffered from enteric fever and some small ulcers on the right conjunctiva which soon got well. In 1915 he had suffered from retinal hæmorrhage, at first slight in the right eye and later on hæmorrhage had recurred on several occasions in the left eye, causing it to become almost blind. In 1917 he had suffered from severe hæmorrhage in the left eye causing some retinal detachment. The eye had been practically blind from that time until 1920, when he first consulted Dr. Edgar Browne for trouble in the right eye. According to Dr. Browne's notes the left eye had manifested old hæmorrhage, a detached retina and almost complete absence of vision. Vision in the right eye had been $\frac{6}{9}$ in the centre with some old hæmorrhage in the periphery. According to a note made on August 23, 1921, no light reflex had been present in the left eye.

When the patient came under Dr. D'Arcy's care in March, 1924, he had stated that the left eye was practically blind and had been so for years and that repeated small hæmorrhages in the right eye were causing anxiety; he feared total blindness. Signs in the chest indicative of fibrotic changes had been found, but nothing to indicate any active disease had been discovered. He had reacted to a subcutaneous tuberculin test and had been treated from April 1, 1924 (P.T.O. 0·005 cubic centimetre), until October 4 of the same year (P.T.O. 0·035 cubic centimetre). He had not lost one day's work during the whole of this period, but treatment was stopped on account of rather severe and repeated febrile reactions. He had then been referred back to Dr. Browne who reported that an extra-

ordinary change had occurred. In the left eye in which for years there had been no light reflex and in which no details of the fundus had been discernible, it was possible to get a good view of the fundus and the retinal detachment had disappeared. Dr. Browne had reported that black specks only could be seen in the fundus of the right eye and that in the left eye fine threads and what looked like parts of an old detachment could be seen. The vision in both eyes was $^6/_9$ partially with correction. Dr. Cowan said that the patient had not come to him professionally since 1924, but he gathered from Dr. Browne's notes that the patient had had a few minor disturbances during 1925, but still retained good vision with correction. During the tuberculin treatment no local treatment had been given to the eye, but several bad teeth had been extracted during this period owing to pathological conditions around the roots. Obviously this might have been a factor in the extraordinary improvement that had occurred.

Dr. Cowan's second patient was a married man, aged forty-one years, who had been referred to him by Dr. Jay in December, 1923. His history was one of recurring vitreous hæmorrhages over a period of eighteen months, the hæmorrhages becoming alarmingly frequent and severe. Both eyes had been infected in turn and after a hæmorrhage vision in the affected eye had been reduced to perception of hand movments. When he first came to see Dr. Cowan, vision in the right eye had been practically nil and in the left eye it had been $^6/_{18}$. The only point of interest in his previous history had been an attack of hæmaturia eight years and again four months previously. Dr. Cowan thought that this might have been of some significance in light of subsequent events. The patient had reacted strongly to tuberculin and he had been treated with tuberculin from January, 1924 (P.T.O. 0·001 cubic centimetre), until May of the same year (O.T. 0·5 cubic centimetre). Except for a few minor disturbances at the beginning of treatment, no further hæmorrhages had occurred. Dr. Jay had reported in June, 1924, that a thick vertical opacity was present in the right eye and that the vision with correction was $^6/_9$. Fine opacities like dust had been present in the left eye and the vision had been $^6/_9$ with correction. This patient had had no other treatment whatever during the time that he was having tuberculin. He had not lost one day's work and said that his vision was perfect.

Dr. Cowan's next patient was a single girl, aged nineteen years. Dr. Cowan said that the patient was suffering from pulmonary tuberculosis and he was showing her to illustrate an error which was deplorably common, so common as to be almost the rule. It was an error which the profession could and should avoid and until they learned to do so, no progress would be made in the management of this all too common disease.

Up to the age of thirteen the patient had been a healthy happy child. Following influenza she had a period of ill health for three years, the main symptoms being irritability and tiredness. The change had been obvious to her mother who had her examined by three different doctors and was told there was nothing to worry about, her symptoms being due to approaching menstruation. In Dr. Cowan's opinion this was pernicious teaching; menstruation was a physiological function and medical men should not suggest that its onset was accompanied by symptoms such as these.

The real cause of her ill health had been disclosed at the end of three years when she had a severe hæmoptysis. Se had had a hard battle since and was at present being treated by artificial pneumothorax combined with good living and specific therapy in the form of subcutaneous injections of tuberculin.

A tuberculin test would have saved all this trouble, as the disease would have been recognized at a stage when it was still confined by the lymphatic system and cure a matter of comparative ease and certainty.

The fourth patient was a girl, aged thirteen years. Her history was interesting. Up to the age of ten she had been a healthy happy child. Following a fall at school she had suffered from pains in the region of the right hip and after about six weeks she had to take to bed. She had been found to have inflamed glands in the groin which were fomented. She was in her bed for two months and away from school for six months. Three or four months after her return to school, during which time she was not well, she had had to go to bed again with enlarged glands in both groins. After a few weeks' rest these had disappeared, but she developed a cough.

About this time she had been taken to a city physician who on the evidence of clinical and X ray examination told the mother that the child was suffering from tuberculous bronchial glands. She was kept away from school and treated for two years. In her mother's words: "A small fortune was spent in medicine, but she seemed no better." Her chief symptoms were tiredness, irritability, cough, loss of appetite, fleeting pains and a leucorrhœal discharge which had been explained to the mother as being due to the child's poor general health.

The patient had come into Dr. Cowan's hands about a month prior to the meeting. She had definite enlarged root glands and this was confirmed by the X ray picture (produced). No reaction whatever had been obtained to tuberculin tests either von Pirquet or subcutaneous. In consultation with Dr. Wunderly active tuberculosis had been excluded. There had been no evidence to suggest Hodgkin's disease. The blood picture was normal and the Wassermann test yielded no reaction.

Against the mother's wish he had insisted that the leucorrhœa should be investigated and a smear disclosed the presence of the gonococcus and this finding was confirmed a week later.

Dr. Cowan thought that he might be excused a certain mild elation over the case. Without a tuberculin test exactly the same blunder would have been committed. The mother had been aware of the presence of this discharge for over two and a half years and there was no reasonable doubt that the original adenitis was an inguinal bubo and all the subsequent symptoms had been due to this infection.

She was making considerable improvement under treatment and it was worth noting that she had almost lost her cough which was stated to be a pronounced symptom, and had had no further rheumatic pains.

The history of Dr. Cowan's next patient was published in The Medical Journal of Australia, October 22, 1921, at page 344. The condition was one in which renal tuberculosis was diagnosed as a result of tuberculin injection. Deposits of lime had occurred and hæmaturia resulted. Dr. Cowan said that tuberculosis in a kidney was curable just as in any other part and the tendency to remove every kidney in which there was a tuberculous deposit, was to be strongly deprecated. Tuberculosis in a kidney was always a secondary process and removal did not eradicate the primary focus. Of course, if the kidney was disorganized and non-functioning, it was better removed, provided always that the other kidney was healthy.

Dr. Cowan's next patient was a married man, aged thirty years, who was suffering from tuberculous ulceration of the bladder. His symptoms dated from Christmas, 1925, and were chiefly pain, frequency of micturition, hæmaturia and pyuria. He had lost about 6·3 kilograms (a stone) in weight. He had had no previous illnesses and there was no tuberculosis in the family or in any of his close associates to his knowledge.

Physical examination disclosed nothing beyond some tenderness in the right iliac fossa and lower part of the abdomen. Rectal examination revealed nothing abnormal. He was in good general condition and his temperature was normal. The urine contained albumin, pus and blood and tubercle bacilli were present. X ray examination revealed both kidneys to be of normal size, shape and position and there was no evidence of urinary calculus.

X ray examination of the chest showed a few enlarged glands in both roots, but no shadows in the outer lung fields or apices to suggest pulmonary tuberculosis. Cystoscopic examination revealed a great deal of bladder involvement, many large ulcers were present around the ureteric orifices and many hæmorrhagic patches. The ureters could not be catheterized and so no evidence as to the condition of his kidneys was available.

The patient had not been considered amenable to surgical treatment and had been referred to Dr. Cowan by Dr. Corbin.

It was proposed to treat him by good living and tuberculin injections. He had already had two injections without any reaction.

Dr. Cowan had had no personal experience of these cases, but the patient's progress would be watched with interest. It was a serious condition, but Dr. Cowan hoped that he would be able to show the patient again at a later date with his ulcers healed.

Dr. Cowan's last patient illustrated a condition which he had not seen before. The patient was a girl, aged fourteen years, who had previously been shown by Dr. Helen Mayo at the Children's Hospital as suffering from chronic lung infection, not tuberculous.

She had recently come under Dr. Cowan's care in the Adelaide Hospital. She had cough and expectoration and some physical signs at the posterior bases. The first specimen of sputum sent to the laboratory had been returned with the report that it contained tubercle bacilli. By pure chance it had been suggested from the laboratory, that it would be wise to get confirmation of this finding. Every subsequent specimen sent down had contained no tubercle bacilli. She had been tested with tuberculin by the subcutaneous test and gave no reaction whatever.

Dr. Cowan said that despite the first sputum report the condition was confidently believed to be non-tuberculous. Both her antra had been opened through the nose, were draining well and were being irrigated daily. Despite her chronic lung infection, she was in good condition and was captain of her basket ball team.

The explanation of the first sputum report was left in other hands, although it was possible that there were such things as tubercle bacillus carriers who harboured the germs in their naso-pharynges.

Dr. H. S. Newland, C.B.E., D.S.O., read a paper entitled "The Surgical Treatment of Tuberculosis" (see page 551).

MEDICO-POLITICAL.

Transactions of the Council of the Victorian Branch.

The following are the most important transactions of the Council of the Victorian Branch of the British Medical Association during the last three months.

New Members.

Fifty-seven new members have been elected.

Big Brother Movement.

Dr. W. Kent Hughes was appointed a representative of the Council on the Big Brother Movement.

Hydatid Disease.

The Council has offered its cooperation with the medical officers of health in combating hydatid disease.

Meetings of Subdivisions.

The Council decided on a scheme of subdivisional meetings during July and August. These Meetings were well attended, when matters of local and general interest were discussed and representatives on the Council for 1927 were elected.

A divisional meeting at Geelong was arranged for July 21, 1926, when a clinical meeting was held and papers were read. The visiting members were also entertained at dinner and motor car drives on the Sunday.

A similar meeting has been arranged for Bendigo on October 23, 1926.

An invitation was received from the Mildura Council for members of the Branch to spend a week in Mildura as its guests. It was hoped that arrangements could be made to make this fit in with a local subdivisional meeting, but it was found that the programme for the balance of the year was well filled already and the invitation for 1926 was reluctantly declined.

Broadcasting.

The Council was of opinion that it was desirable that all public pronouncements to be broadcast on medical matters should be made anonymously by a member or members approved by the Council.

Five matters proposed to be broadcast were referred to the Council by the Manager of 3LO for its approval and in each case its opinion was respected.

Notification of Deaths.

It was learnt from the Chief Secretary's Office that under the act a medical practitioner in attendance on a patient must notify the Registrar of Births, Deaths and Marriages within twenty-one days of the death. The responsibility does not rest on the undertaker who usually supplies such information.

Welfare of Women and Children.

At the request of the Council 1,250 copies of the Report of Welfare of Women and Children by Drs. Mann and Scantlebury were sent gratis from the Public Health Department to each member of the medical profession in Victoria.

The Public Health Department asked for an opinion of the Council as to notification of a birth within three days, the object being to enable the infant welfare authorities to get into early touch with the mother. A reply was sent that as the notification would result in an infant welfare nurse visiting a house where a family doctor was in attendance, the Council did not agree to the proposal.

Homœopaths.

It was resolved to abolish the homœopathic list.

Letting of Hall.

For use of the hall and council chamber it was resolved to charge a nominal fee to sections and kindred associations for cost of lighting and cleaning.

Friendly Society Lodge Guarantee Fund.

The balance of money in hand from the guarantee fund raised during the lodge dispute some years ago was returned *pro rata* to the contributors.

Industrial Medical Officers.

The following rates of pay for industrial medical officers have been approved: One guinea per attendance of one hour or fraction of an hour per day, under Section 3 of the industrial medical officers' agreement (approved by the Federal Committee) shall be paid. Visits made to the factory outside the hours specified in the agreement shall be charged for at ordinary fees. Where more frequent attendances are required, the minimum salary that shall be paid, shall be subject to the approval of the Council. Where the attendance is required on each day of the week, a miniumm annual salary of £250 shall be paid.

It was recommended that a health officer making Schick tests should charge one guinea per hour in addition to travelling expenses.

Remuneration of Municipal Health Officers.

With regard to payments to municipal health officers the Council resolved that extra work should be charged for at the rate of one guinea per hour and travelling expenses. The duties of health officers should be those laid down in the general code under the *Health Act*, 1919, and the salary as recommended by the conference of medical officers of health, 1919.

Hospitals for Epidemic Diseases.

It was resolved that epidemic hospitals should be provided.

Lodge Agreements.

At the instance of the Federal Committee, the Council approved of the principle of a uniform lodge agreement for the whole of the Commonwealth.

Library Arrangements.

The Public Library authorities were asked by a general meeting of members and by the Council to throw open

to medical practitioners and students that portion of the library dealing with medicine and allied diseases. A reply was received that none of the medical literature was recent and was on the stack shelves in the basement and so the request could not be complied with. Current periodicals were available on request.

Ethical Matters.

An ex-assistant to a medical practitioner was informed that he should not put up his plate or have his consulting rooms within three miles of the residence of his late principal, but he might see any patients residing within that proscribed area, provided he had not seen them professionally as assistant.

A suburban medical practitioner had been offered a position as medical attendant to all employees of a factory on a condition that he accepted a fee smaller than that of the standard scale of fees. The Council informed him that it was not in favour of the proposal.

It was decided that before election each member should be provided with a copy of the ethical rules.

Publicity.

A public questions committee was formed and its first report was adopted.

Its activities were defined as being to give information to the press on matters of interest which might be brought before the public in cable messages as any other way, when asked by the press to do so, such information to be authentic; secondly, to instruct and educate the public by means of articles in the press or some other way on matters which the committee thought the public should be informed about by preventive medicine, tuberculosis *et cetera*. The editors of the leading newspapers approved of the proposal and it was agreed that anonymity should be preserved. Presidents and secretaries of the various sections have been asked to give the required information themselves or to put the editors on to the best man for the purpose. The Colonial Mutual Life Association will publish a pamphlet on cancer in cooperation with this committee.

With regard to the notification in the lay newspapers of attendance by practitioners upon patients, the Council advises members that it has repeatedly requested the editors of the daily newspapers not to publish the names of medical practitioners and that the editors have repeatedly refused to withhold such names in special cases, such as vice-royalty.

NOMINATIONS AND ELECTIONS.

The undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Frew, Charles Alexander, M.B., Ch.M., 1923 (Univ. Sydney), 189, Macquarie Street, Sydney.
Abramovich, Leslie, M.B., Ch.M., 1925 (Univ. Sydney), The Bungalow, Ethane Avenue, Randwick.
Horniman, Robert Vicary, M.B., Ch.M., 1926 (Univ. Sydney), Fairlight Street, Manly.
Lawson, Colin Alexander, M.B., Ch.M., 1925 (Univ. Sydney), Rutledge Street, Eastwood.
Schuch, Robert William, L.R.C.S., L.R.C.P. (Edinburgh), 1926, L.F.P.S. (Glasgow), 1926, Anderson Street, Chatswood.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Arnott, Desmond William Holme, M.B., Ch.M., 1925 (Univ. Sydney), Psychiatric Clinic, Broughton Hall.
Balzer, John Camfield, M.B., Ch.M., 1926 (Univ. Sydney), Mater Misericordiæ Hospital, North Sydney.
Davis, Keith Joseph Brandon, M.B., Ch.M., 1926 (Univ. Sydney), 34, Gowrie Avenue, Waverley.
Dickson, Ian Thomas, M.B., Ch.M., 1926 (Univ. Sydney), Rabaul, New Guinea.
Goode, Caleb James Frew, M.B., Ch.M., 1926 (Univ. Sydney), 99, New Canterbury Road, Petersham.
Laws, Brenda Elizabeth Charles, M.B., Ch.M., 1925 (Univ. Sydney), Grasmere, Albyn Road, Strathfield.
Miles, Thomas William, M.B., Ch.M., 1926 (Univ. Sydney), Tallimba, New South Wales.
Oag, Hugh Stewart, M.B., Ch.M., 1925 (Univ. Sydney), Campbell Street, Hunter's Hill.
Peck, Grace Jean, M.B., Ch.M., 1926 (Univ. Sydney), Henry Street, Gordon.
Price, Alexander Vivian Gordon, M.B., 1926 (Univ. Sydney), Gizo, British Solomon Islands Protectorate.
Vickers, Allan Robert Stanley, M.B., Ch.M., 1926 (Univ. Sydney), 47, Burwood Road, Belmore.
Wheelihan, John Maurice, M.B., Ch.M., 1926 (Univ. Sydney), Clyro, Victoria Road, Bellevue Hill.
Wilkinson, Frederick Osborne Bushby, M.B., 1926 (Univ. Sydney), Cranbrook Road, Rose Bay.

Public Health.

REPORT OF THE ROYAL COMMISSION ON HEALTH.

Conference of Ministers of Health.

A conference of the Ministers of Health of the Commonwealth and of the several States was held at Melbourne on July 21 and 22, 1926, to discuss the recommendations included in the report of the Royal Commission on Health. The Commonwealth was represented by the Honourable Sir Neville Howse, V.C., K.C.B., K.C.M.G., Minister of Health, Dr. J. H. L. Cumpston, Director-General of Health, the Honourable George Cann, Minister of Health of New South Wales, Dr. Robert Dick, Director-General of Public Health of New South Wales, the Honourable Dr. Stanley S. Argyle, Minister of Health of Victoria, Dr. E. Robertson, Chairman of the Health Commission of Victoria, the Honourable J. Stopford, Minister of Health of Queensland, Mr. C. B. Chuter, Assistant Uunder Secretary of Queensland, the Honourable James Jelley, Minister of Health of South Australia, Dr. W. Ramsay Smith, President of the Central Board of Health of South Australia, the Honourable S. W. Munsie, Minister of Health of Western Australia, Dr. John Dale, Deputy Commissioner of Public Health of Western Australia, Mr. E. J. Tudor, Secretary of the Department of Public Health, representing the Minister of Health of Tasmania.

The following resolutions were adopted by the members of the conference.

Part I. of Report.—Morbidity.

1. The conference agrees that an early conference should be held of the professional heads of the Health Departments, followed by a conference of these with the statisticians (Commonwealth and State), consulting later with representative hospital superintendents.

The Commonwealth will convene these conferences and bear the expenses of the delegates.

These conferences should improve the existing system of collecting mortality statistics, extend the collection of morbidity statistics and remove any existing anomalies.

Part III. of Report.—Cooperation of Commonwealth and State Health Authorities.

2. The conference recommends that the Commonwealth take steps with regard to a school of preventive medicine and tropical hygiene, to consult the various medical schools and universities of the State with a view to the establishment of such a school and to the teaching of preventive medicine on an improved basis to all medical students and other public health personnel.

3. The conference agrees that a federal health council should be established as an advisory body. The conference

further agrees that the recommendations of the Royal Commission on Health are in general accepted and should be adopted by the Federal Health Council as the general policy to be followed.

The functions of the Federal Health Council should be as specified by the Royal Commission.

The Commonwealth Government will bear all the expenses of this council including the expenses of delegates.

The Federal Health Council will meet at least annually.

The Commonwealth Director-General of Health will preside at all sessions of the Federal Health Council.

Part IV. of Report.—Prevention of Disease.

4. The conference affirms the general principle that all Government services connected with health should be under the control of one minister.

5. The conference agrees to refer the question of the model scheme of general health administration to the Federal Health Council.

6. The Commonwealth Government after recommendation by the Federal Health Council may formulate the principles of a comprehensive campaign as the basis for any subsidies the Commonwealth may make to the States for carrying out such a campaign.

7. The conference agrees to refer the question of laboratories to the Federal Health Council.

8. The conference agrees with Recommendations 6 and 7 of Part IV. of the Royal Commission's Report.

Part V. of Report.—Venereal Diseases.

9. The conference approves generally of the recommendations in Part V. (venereal diseases), without expressing an opinion on Clause 13.

Part VI. of Report.—Food and Drugs.

10. The matter of the control of imported foods and such foods and drugs of Australian origin as are or may be the subject of interstate trade shall be referred to the Federal Health Council, together with analysts and trade representatives, on the lines adopted in the constitution of the conferences of 1910 and 1913, with a view to recommendations being made to the Commonwealth Government and the Governments of the various States in relation to the relative spheres of administration of each.

Part VII. of Report.—Maternity Hygiene.

11. The Commonwealth Government after recommendation by the Federal Health Council may formulate standards for facilities to women before, during and after childbirth, as the basis for any subsidies the Commonwealth may make to the States for the provision of such facilities.

12. That Recommendations 3 and 4 of Part VII. be referred to the Federal Health Council.

Part VIII. of Report.—Child Welfare.

13. The Commonwealth Government, after recommendation by the Federal Health Council, may formulate standards for facilities for child welfare, especially schools of mothercraft and also to provide institutional care for mental defectives and bush nursing as the basis for any subsidies the Commonwealth may make to the States for the provision of such facilities.

14. Recommendation 4 adopted.

Part IX. of Report.—Industrial Hygiene.

15. The conference endorses the general principles of Part IX., referring the details to the Federal Health Council.

Part X. of Report.—Research Work.

16. The recommendations in Part X. relative to the encouragement and development of research work were adopted unanimously.

17. The conference expresses the opinion that the Commonwealth Department of Health should investigate questions of diet in relation to health and disease and keep the State Departments informed of the results obtained.

Part XI. of Report.—Relationship Between Public Health Authorities and Medical Practitioners.

18. The conference endorses generally Recommendation 3 of Part XI. and refers the consideration of details to the Federal Health Council.

The same course should be followed in respect of dentists, nurses and midwives.

Part XIII. of Report.—Publication of Information Relating to Public Health.

19. The conference adopts the recommendations of the Royal Commission with reference to Part XIII.

The Honourable J. Stopford (Queensland) referred to the following motion of which he had previously given notice. This, although not moved as a definite resolution, was accepted by the conference:

> That it is eminently desirable that the Commonwealth should undertake research work and special investigations and campaigns as in the case of hookworm, compilation of statistics and publicity and for these purposes the States should cooperate and extend to Commonwealth officers the powers and authorities of the State laws as and when required and enable access to and make available the records and resources of State institutions and activities.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

The Executive Committee of the second session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927, has issued the following provisional programme of work of the sections. It should be recognized that this programme is in outline only. Additions and emendations may be made at a later date.

General.

There will be four full working days, February 3, 6, 7 and 8, 1927. On the mornings of these days before the commencement of the sectional sessions there will be clinical demonstrations in hospitals and other institutions.

The morning sessions will start at 9.45 a.m. and close at 12.45 p.m.; the afternoon sessions will start at 2.15 p.m. and close at 4.15 p.m.

The work will be carried out in twelve sections. Each section has a president from one of the States of Australia and a number of vice-presidents from the Branches in Australia and New Zealand. The members of the committees of the several sections and the honorary secretaries of sections are members resident in Dunedin.

Sectional Meetings.

Combined Meetings.

Friday, February 3, 1927.—Morning Session.

Section I. (Medicine), Section II. (Surgery), Section IV. (Pathology and Bacteriology), Section V. (Preventive Medicine), Section XII. (Radiology).—"Goitre."

Tuesday, February 7, 1927.—Morning Session.

Section III. (Obstetrics and Gynæcology), Section V. (Preventive Medicine), Section IX. (Pædiatrics).—"The Prevention of Disease in Infancy and Childhood."

Afternoon Session.

Section II. (Surgery), Section IV. (Pathology and Bacteriology), Section V. (Preventive Medicine).—"Parasitology; Hydatid Tests of Clinical Interest." "Hookworm in Australia and Samoa."

Section XI. (Orthopædics), Section X (Naval and Military Medicine and Surgery).—"The Cripple in War and in Peace."

Wednesday, February 8, 1927.—Morning Session.

Section I. (Medicine), Section II. (Surgery), Section VIII. (Neurology), Section XI. (Orthopædics).—"Spastic Paraplegia."

All Sections.—"Cancer; an Outline of the Present Position" (from 11.30 a.m.).

Individual Sections.

Section I.—Medicine.

February 3, 1927.—Afternoon session: "Goître" (papers).
February 6, 1927.—Morning session: "Diet and Disease" (discussion). Afternoon session: "Renal Conditions."
February 7, 1927.—Morning session: "Medical Aspects of Hydatid Disease." Afternoon session: "Blood Conditions."
February 8, 1927.—Afternoon session: Meeting with the Section of Neurology.

Section II.—Surgery.

February 3, 1927.—Afternoon session: President's address on goître; other papers on special aspects of goître.
February 6, 1927.—Morning session: Papers on alimentary conditions and discussion. Afternoon session: Papers on urology and discussion.
February 7, 1927.—Morning session: Papers on Raynaud's disease and on splenectomy in blood diseases. Afternoon session: "The Surgical Aspects of Hydatid Disease."
February 8, 1927.—Afternoon session: Meeting with the Sections of Pathology and Bacteriology and of Radiology; "Inflammations and Tumours of Bone."

Section III.—Obstetrics and Gynæcology.

February 3, 1927.—Afternoon session: President's address and papers on prolapse.
February 6, 1927.—Morning session: "The Problem of Cæsarean Section" (discussion). Afternoon session: Papers on Rubin's apparatus.
February 7, 1927.—Afternoon session: Paper on venereal disease work.
February 8, 1927.—Morning session: Papers. Afternoon session: Paper and discussion on puerperal sepsis.

Section IV.—Pathology and Bacteriology.

February 3, 1927.—Afternoon session: Papers on goître.
February 6, 1927.—Morning session: Papers on renal conditions and on hæmatology. Afternoon session: "Poliomyelitis" (paper and discussion).
February 7, 1927.—Morning session: Papers on serological work; comparative pathology. Afternoon session: Papers on tropical conditions.
February 8, 1927.—Morning session: "Differential Action of X Rays." Afternoon session: "Inflammations and Tumours of Bone."

Section V.—Preventive Medicine.

February 3, 1927.—Afternoon session: Papers on goître.
February 6, 1927.—Morning session: "Diet and Nutrition." Afternoon session: "Diet and Nutrition."
February 7, 1927.—Morning session: Combined meeting. Afternoon session: "Parasitology" (papers).
February 8, 1927.—Morning session: Papers on forecasting of outbreak of diphtheria.
February 9, 1927.—Afternoon session: "Industrial Hygiene" (papers and discussion).

Section VI.—Ophthalmology.

This Section will meet only in the mornings.
February 3, 1927.—President's address. "The Cause of Concomitant Strabismus" (paper on the ocular signs of goître).
February 4, 1927.—Combined meeting with Section VII. "Asthenopia and Nasal Insufficiency."
February 6, 1927.—"Treatment of Non-Suppurative Intraocular Infections." "The Uses of Red-free Light in Ophthalmology."
February 7, 1927.—"Treatment of Strabismus." "The Teaching of Ophthalmology for Medical Students."
February 8, 1927.—"Malaria Treatment of Optic Atrophy from Syphilis." "Retinitis Pigmentosa." "Ontophoresis in Ophthalmic Work."

Section VII.—Otology, Rhinology and Laryngology.

This Section will meet only in the afternoons.
February 3, 1927.—"Otoneurology and the Diagnosis of Intracranial Disease." "Paralysis of the Laryngeal Nerves in Goître."
February 4, 1927.—Combined meeting with Section VI. "Asthenopia and Nasal Insufficiency."
February 6, 1927.—Papers and discussion on sphenoethmoidal disease. "Vaccines in Diseases of the Nose."
February 7, 1927.—"Operative Treatment of Disease of the Mastoid." "Conservatism in Treatment of Middle Ear Disease. Acute *Otitis Media*."
February 8, 1927.—"Sinusitis in Children." "Allergy."

Section VIII.—Neurology.

This Section will meet only in the afternoon, except on February 8.
February 3, 1927.—President's address. Papers on the physical basis for mental disorder.
February 6, 1927.—"The Social Aspect of Mental Disorder." "University Teaching of Psychiatry."
February 7, 1927.—"The Voluntary Patient." "Delinquency."
February 8, 1927.—Morning session: Combined session with Sections I., II. and XI. Afternoon session: "Sequelæ of Lethargic Encephalitis." "Localization of Spinal Tumours." Cinema demonstration of the teaching of neurology.

Section IX.—Pædiatrics.

February 3, 1927.—Afternoon session: President's address. Papers on infant feeding.
February 6, 1927.—Morning session: "Rheumatic Heart Disease." Afternoon session: "Rheumatic Heart Disease."
February 7, 1927.—Afternoon session: Visit to the Karitane Hospital (papers).
February 8, 1927.—"Erythrœdema."

Section X.—Naval and Military Medicine and Surgery.

February 3, 1927.—Morning session: Papers. Afternoon session: Papers.
February 4, 1927.—Staff ride.
February 6, 1927.—Morning session: Discussion on the staff ride. Afternoon session: Combined session with Section XI.
February 7, 1927.—Morning session: Papers. Afternoon session: Papers.
February 8, 1927.—Morning session: Papers.

Section XI.—Orthopædics.

This Section will meet in the afternoons only.
February 3, 1927.—President's address. "Muscle Tone."
February 6, 1927.—Combined meeting with Section X.
February 7, 1927.—Papers.
February 8, 1927.—Combined meeting with Sections I., II. and VIII.

Section XII.—Radiology.

February 3, 1927.—Morning session: Combined meeting on goître. Afternoon session: Papers.
February 6, 1927.—Morning session: President's address. Papers on the alimentary tract.

February 7, 1927.—Morning session: Papers. Afternoon session: "Hydatid Work," with Section II.

February 8, 1927.—Morning session: "X Ray Treatment of Cancer." Afternoon session: "Bone Conditions."

It is hoped that there will be a general meeting in the evening of one of the days at which the relationship of the State to the practitioner will be discussed.

Correspondence.

MENDEL AND GENIUS.

SIR: In an article on "Mendel and Genius" in THE MEDICAL JOURNAL OF AUSTRALIA, October 2, 1926, Dr. Taylor writes: "Mendel was a priest with few of the advantages which the modern scientist possesses and he has given a wonderful gift to life, the irony of which is that its practical application is destructive in a degree to some of the tenets of his faith."

Religion and science can in no way be opposed and therefore I think Dr. Taylor is in error in what he believes to be the tenets of the faith of Mendel.

I would therefore be obliged if Dr. Taylor would state in what way he believes the practical application of Mendel's discoveries to be destructive to the tenets of his faith.

Yours, etc.,

H. B. OXENHAM, M.B., Ch.M. (Sydney), L.M.R.C.P.I.

Leichhardt, Sydney,
October 5, 1926.

A CHAIR OF PREVENTIVE MEDICINE.

SIR: In reply to Dr. Purdy may I make it perfectly clear that I am an enthusiastic supporter of the teaching of preventive medicine, but I have a decided objection to the creation of a chair of preventive medicine, because I believe the proper road to success does not lie in that direction. If those interested will look up any medical textbook and turn to the account of any disease, they will find under certain formal heading a number of indications, such as

Definition,
Cause,
Diagnosis,
Prognosis,
Treatment *et cetera*.

Why should there not be added in every instance two further sections:

Mode of Prevention,
Disabilities Resulting from the Diseases Indicated.

If then in the lectures given in the various medical schools these two sections were emphasized, the medical student would receive training in preventive medicine.

I am of opinion that preventive medicine will not make the headway it should, no matter how skilful may be the public health staff, until the general practitioner takes a hand in the matter. The advice tendered by the general practitioner to the patient and his family is the basis of proper medical progress. Unless as a student he has been trained in the manner indicated, progress must necessarily be slow.

I have nothing except praise to say for the proposal to create a proper school of public health in which officers may be efficiently trained. My objection is solely to the attempt to erect preventive medicine into a subject for special training instead of regarding it as an attitude of mind which should be inculcated into every medical student.

Yours, etc.,

JAMES W. BARRETT.

105, Collins Street, Melbourne,
October 6, 1926.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act*, 1912 and 1915, as duly qualified medical practitioners:

Abbott, Joseph Henry, M.B., 1926 (Univ. Sydney), 69, Liverpool Road, Summer Hill.

Abdullah, Anthony Dominic, M.B., 1926 (Univ. Sydney), 97B, Auburn Road, Auburn.

Anderson, Leighton Rowland, M.B., 1926 (Univ. Sydney), Newinga, Moruben Road, Mosman.

Baker, Joseph Roy, M.B., 1926 (Univ. Sydney), Brixton, 34, Marian Street, Enmore.

Black, John Roland, M.B., 1926 (Univ. Sydney), Hopetoun Avenue, Vaucluse.

Bolger, Thomas Natal, M.B., 1926 (Univ. Sydney), 2, Wentworth Street, Point Piper.

Collins, Kevin James, M.B., 1926 (Univ. Sydney), 117, Queen Street, Woollahra.

Croft, Roy Wood, M.B., 1926 (Univ. Sydney), 165, High Street, North Sydney.

Forster, Cameron McDougall, M.B., 1926 (Univ. Sydney), Avalon, Arthur Street, Edgecliff.

Geaney, Norman, M.B., 1926 (Univ. Sydney), Howard Street, Baroona Heights, Brisbane, Queensland.

Graham, Colin Spencer, M.B., 1926 (Univ. Sydney), Lane Cove Road, Turramurra.

Healy, Jean Florence Hamilton, M.B., 1926 (Univ. Sydney), Oakleigh Court, Dalley Street, Waverley.

Horniman, Robert Vicary, M.B., 1926 (Univ. Sydney), Lufru, Fairlight Street, Manly.

Hughes, Norman William Michael, M.B., 1926 (Univ. Sydney), 20, Cook Road, Centennial Park.

James, Stanley George, M.B., 1926 (Univ. Sydney), 47, Barry Street, Neutral Bay.

Little, Harry Bennett, M.B., 1926 (Univ. Sydney), Gordon Road, Roseville.

Longworth, Roland Edward, M.B., 1926 (Univ. Sydney), Craig Naw, Prince Edward Parade, Hunter's Hill.

McHardy, Charles Alister, M.B., 1926 (Univ. Sydney), 24, Kulgoa Road, Bellevue Hill.

Marshman, Eric Ambrose Claude, M.B., 1926 (Univ. Sydney), 43, Second Street, Canterbury.

Martell, Alexander Mayne, M.B., 1926 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

O'Brien, Clifford Raymond, M.B., 1926 (Univ. Sydney), 2, Carden Flats, Coogee.

Peck, Grace Jean, M.B., 1926 (Univ. Sydney), Cooinoo, Henry Street, Gordon.

Potiris, Michael, M.B., 1926 (Univ. Sydney), 45, Cook Road, Centennial Park.

Punch, Francis Michael Greenway, M.B., 1926 (Univ. Sydney), Ronaele, Julia Street, Ashfield.

Robertson, Fanny Croaker, M.B., 1926 (Univ. Sydney), Rafmestham, Strickland Avenue, Roseville.

Ryan, Edward Joseph, M.B., 1926 (Univ. Sydney), 21, Holborrow Street, Croydon.

Shannon, James Robert, M.B., 1926 (Univ. Sydney), c.o. C. S. Hungerford, Standonnel, Wentworthville.

Shappere, Arthur Joseph, M.B., 1926 (Univ. Sydney), Walma'een, Hilltop Crescent, Manly.

Spark, Robin James Hester, M.B., 1926 (Univ. Sydney), Wallscourt, Peat's Ferry Road, Hornsby.

Taylor, Hugh Carlyle, M.B., 1926 (Univ. Sydney), Turanville, Scone.

Thomson, Vida Mary, M.B., 1926 (Univ. Sydney), Supreme Court Hotel, King Street, Sydney.

Walker, Jack Frederick, M.B., 1926 (Univ. Sydney), Wilga, Cressy Street, Canterbury.

Wheelihan, John Maurice, M.B., 1926 (Univ. Sydney), Clyro, Victoria Road, Woollahra.

Wilkinson, Frederick Osborne Bushby, M.B., 1926 (Univ. Sydney), Enwoola, Cranbrook Road, Rose Bay.

Williams, Gwyneth Elizabeth, M.B., 1926 (Univ. Sydney), Durham, Queen Victoria Road, Drummoyne.

For Additional Registration.

Morris, Emanuel Sydney, M.D., 1926 (Univ. Sydney), Victoria Square, Ashfield.

Windeyer, John Cadell, M.D., 1926 (Univ. Sydney), Water Street, Wahroonga.

Books Received.

PATHOLOGY AND TREATMENT OF THE INFLAMMATORY DISEASES OF THE NASAL ACCESSORY SINUSES, by Professor Dr. M. Hajek, Translated and Edited by Joseph D. Heitger, A.B., M.D., and French K. Hansel, M.D., M.S.; Fifth Edition; Volumes I. and II.; 1926. St. Louis: The C.V. Mosby Company. Royal 8vo., pp. 724, with illustrations. Price: $17.00 each net.

ELECTROTHERMIC METHODS IN THE TREATMENT OF NEOPLASTIC DISEASES, by J. Douglas Morgan, B.A., M.D.; 1926. Philadelphia: F. A. Davis Company. Post 8vo., pp. 172, with illustrations. Price: $2.50 net.

ELEMENTS OF PATHOLOGY, by Aller G. Ellis, M.Sc., M.D.; 1926. Philadelphia: P. Blakiston's Son and Company. Royal 8vo., pp. 554, with illustrations.

CAVERNOUS SINUS THROMBOPHLEBITIS AND ALLIED SEPTIC AND TRAUMATIC LESIONS OF THE BASAL VENOUS SINUSES, by Wells P. Eagleton, M.D.; 1926. New York: The Macmillan Company. Post 8vo., pp. 211.

FUNDAMENTALS OF DERMATOLOGY, by Alfred Schalek, M.D.; 1926. Philadelphia: Lea and Febiger. Post 8vo., pp. 250, with illustrations. Price: $3.00 net.

THE DISEASES OF WOMEN: A HANDBOOK FOR STUDENTS AND PRACTITIONERS, by Sir John Bland-Sutton, Bart., F.R.C.S. (England), LL.D., and Arthur E. Giles, M.D., B.Sc. (London), F.R.C.S. (Edinburgh); Eighth Edition; 1926. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 610, with illustrations. Price: 15s. net.

AURICULAR FIBRILLATION, by J. G. Emanuel, B.Sc., M.D., F.R.C.P.; 1926. Birmingham: Cornish Brothers Limited. Royal 8vo., pp. 31. Price: 3s. 6d. net.

THE INFLAMMATORY AND TOXIC DISEASES OF BONE: A TEXT-BOOK FOR SENIOR STUDENTS, by R. Lawford Knaggs, M.C. (Cantab.), F.R.C.S., with photomicrographs by G. H. Rodman, M.D., Hon. F.R.P.S.; 1926. Bristol: John Wright and Sons Limited. Royal 8vo., pp. 428. Price: 20s. net.

Medical Appointments.

Dr. Stuart Kay (B.M.A.) has been appointed Quarantine Officer, at Mackay, Queensland.

* * * *

Dr. Richard Sanders Rogers (B.M.A.) has been appointed a Member of the Board of Governors of the Public Library, Museum and Art Gallery of South Australia.

* * * *

Dr. Ida Gertrude Margaret Halley (B.M.A.) and Dr. John Smith Proctor (B.M.A.) have been appointed Official Visitors to the Mental Hospital, Parkside, South Australia.

* * * *

Dr. Esmond Frank West (B.M.A.) has been appointed Honorary Anæsthetist to the Adelaide Hospital.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants *locum tenentes* sought, etc., see "Advertiser," page xxiv.

COAST HOSPITAL, LITTLE BAY, SYDNEY: Honorary Assistant Surgeon.

NEWINGTON STATE HOSPITAL, SYDNEY: Honorary Ophthalmic Surgeon.

ROOKWOOD STATE HOSPITAL, SYDNEY: Honorary Surgeon, Honorary Dermatologist.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY: Honorary Relieving Assistant Ophthalmic Surgeon.

WATERFALL SANATORIUM, NEW SOUTH WALES: Junior Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

OCT. 23.—Illawarra Suburbs Medical Association, New South Wales.
OCT. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 27.—Victorian Branch, B.M.A.: Council.
OCT. 28.—New South Wales Branch, B.M.A.: Branch (Ordinary).
OCT. 28.—South Australian Branch, B.M.A.: Branch.
NOV. 2.—Tasmanian Branch, B.M.A.: Council.
NOV. 3.—Western Australian Branch, B.M.A.: Council.
NOV. 4.—South Australian Branch, B.M.A.: Council.
NOV. 4.—Section of Orthopædics, New South Wales Branch, B.M.A.
NOV. 5.—Queensland Branch, B.M.A.: Branch.
NOV. 9.—Tasmanian Branch, B.M.A.: Branch.
NOV. 9.—New South Wales Branch, B.M.A.: Ethics Committee.
NOV. 9.—Section of Medicine, New South Wales Branch, B.M.A.
NOV. 10.—Victorian Branch, B.M.A.: Branch; last date of nominations for Council.
NOV. 11.—Victorian Branch, B.M.A.: Council.

Editorial Notices.

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